

## Highlights of this issue

By Sukhwinder S. Shergill

### Psychosis: non-evidence based psychological and medication management guidelines?

Psychotic illness is one of the most devastating of the mental disorders and all mainstream treatment guidelines advocate the use of medication, psychological treatment and social interventions. However, how robust are the data underlying these guidelines? Two articles in this edition of the *BJPsych* address this question. The first, an editorial, questions the evidence base underpinning recommendations for offering psychological treatments for all patients with psychosis; the second, a review paper, questions the data underpinning the efficacy of quetiapine, one of the most widely used antipsychotic medications. The editorial by Taylor & Perera (pp. 357–259) critiques the recently published National Institute for Health and Care Excellence (NICE) guidelines on the treatment and management of psychosis and schizophrenia, concluding that the promotion of cognitive-behavioural therapy (CBT) in the guidelines goes beyond the available evidence, and in some cases, such as advocating 16 planned sessions, is based on no evidence. They further suggest that the NICE guidelines contain recommendations for pharmacological treatment that are both vague and non-specific, and fail to reflect more recent data on differences in the efficacy of the available antipsychotic medications. The challenge to the efficacy of the antipsychotic quetiapine is made in a review of placebo-controlled trials of the immediate-release form of quetiapine in schizophrenia. Hutton and colleagues (pp. 360–370) conclude that quetiapine treatment has only a small effect on psychotic symptoms over the 2- to 12-week treatment period – demonstrating an improvement of approximately 6.5 points in the total score on the Positive and Negative Syndrome Scale, while a difference of 11 points is proposed as the minimum for patients to feel better, and the analogous number is 15 points difference for raters to notice significant improvement. The NICE guidelines are very highly regarded and quetiapine is a very widely used antipsychotic medication; hopefully, a discussion of the relevant data will make valuable reading in the next correspondence column.

### Post-traumatic stress disorder and personality

What happens to patients after they have suffered physical and psychological trauma? Bryant *et al* (pp. 417–423) followed up over a 1000 patients for up to 6 years after admission to hospital with a traumatic injury. They found five distinct trajectories of patient outcome: 73% were resilient, demonstrating low levels of post-traumatic stress disorder (PTSD) symptoms immediately following the injury and at subsequent follow-up over 6 years; 4% demonstrated chronic high levels of PTSD symptoms, immediately after injury and persisting over the follow-up; and 6% showed a recovery pattern with high levels of symptoms post-injury but reducing in severity over the subsequent 6 years. Interestingly, 10% of the sample demonstrated a worsening pattern, with low levels of symptoms at immediate follow-up but an increase in symptoms over the following 6 years, and a further 8% showed an intermediate worsening but recovery

pattern characterised by low PTSD symptom levels immediately after the injury, increasing PTSD symptoms over the 2 years follow-up but returning to low levels again by the 6-year outcome. The authors report that the poorer-prognosis groups were more likely to have experienced an increased number of life stressors, have suffered mild traumatic brain injury and had an admission to an intensive care unit – these factors may allow more targeted interventions for the high-risk trajectory patients at an earlier phase. An editorial by Newton-Howes *et al* (pp. 355–356) highlights possible disadvantages of dropping the multi-axial classification approach from the DSM-5 and ICD-11 systems, potentially risking a decreased emphasis on personality-related issues, as the specific axis devoted to personality disorders will no longer be present. They agree that personality disorders are not distinct enough from other mental disorders to necessarily warrant a separate axis, but advocate for clinicians to still actively consider mental state and personality disorders in all patients presenting with mental illness – given the importance of the interaction between these diagnoses. In a similar vein, PTSD is often given less prominence when present in a patient with psychotic illness. De Bont and colleagues (pp. 408–416) demonstrated the utility of the Trauma Screening Questionnaire in detecting PTSD in psychosis – with traumatic events noted in 78% of the patients with psychosis, a comparable figure to the general population. However, patients with psychosis had a PTSD prevalence of 16%, relative to 0.5% noted in the clinical record and a general population prevalence of 3.3%. The authors recommend use of this screening instrument, which they have helpfully appended to their paper.

### Depression, genetic variability, and cellular aging

Depression is common and recognised to be a multifactorial illness. Heritability estimates account for over half of the risk, with a brain-derived neurotrophic factor (BDNF) polymorphism being a potential contributor to this increased risk. How this is reflected in neural processing is not clear. MacGregor Legge *et al* (pp. 379–383) show that variations in BDNF polymorphisms affect the structure of key frontal cortical areas, supporting the neurotrophin hypothesis of depression. Increased cortical thinning in the middle frontal cortex was associated with the Met polymorphism in both patients and control participants, while changes in the anterior cingulate were differentially expressed in the depressed group with this specific polymorphism. Ma and colleagues (pp. 385–392) demonstrated that allelic variation in the serotonin transporter gene modulated the effects of the selective serotonin reuptake inhibitor (SSRI) citalopram on neural processing of negative emotional stimuli. The authors suggest that, given that allelic variation is linked to differential response to SSRIs, there may be an option to personalise antidepressant treatment in the future. Anxiety is a risk factor for several age-related illnesses including coronary heart disease and diabetes. Verhoeven *et al* (pp. 371–378) demonstrated that patients with anxiety disorders were more likely to have greater evidence of cellular ageing, as indexed by a shorter leukocyte telomere length, relative to controls. However, this shorter telomere length, compatible with an extra 3–5 years of accelerated ageing, was not observed in patients with remitted anxiety disorder; thus, this process appears to be partly reversible. The authors note that lifestyle changes such as increased physical exercise have had a favourable impact on cellular ageing, and may warrant further investigation.