Highlights of this issue

BY SUKHWINDER S. SHERGILL

EDITORIALS

The National Institute for Clinical excellence (NICE) guidelines on managing depression have been recently released. Whitty & Gilbody (pp. 177–178) reflect on the opportunity offered by their introduction, and suggest that this may be squandered unless significant organisational resource is also allocated to aid their implementation, particularly in primary care. They advocate using new primary mental health workers to bridge the gap between primary and secondary care, perhaps through offering case management to patients in primary care. Considerable technological innovation has led to significant advances in investigating the genetic risk factors for many psychiatric disorders. Farmer *et al* (pp. 179–181) discuss approaches to investigating the genetic and environmental risk factors for affective disorders, particularly emphasising the distinction between a common shared genetic liability between some disorders and other genes being specific for one or other disorder. Similarly, there is increasing interest in the interaction between genes and environment; dissecting out how genes may act to make individuals susceptible to adversity, rather than influencing exposure to adversity per se. This attention to environmental risk factors, clarifying possible mechanisms of interaction, and the evolving pathological phenotype is highlighted as a major focus for research in the post-genomic era. Comorbidity is a widely used term to denote several different coexisting disorders including medical and psychiatric, and more than one psychiatric disorder. Maj (pp. 182–184) asks whether the popularity of the concept of comorbidity is a consequence of the current operationalised diagnostic systems, and questions whether disorders can easily be divided in an artificial manner. In line with this scepticism, Khan *et al* (pp. 190–196) demonstrate that neuroticism, although an unfashionable term, appears to be a broad vulnerability factor for not only depression and anxiety disorders, but also more surprisingly for alcohol and drug dependence and antisocial personality disorder. This ties together issues of comorbidity between disorders and the possibility of common genetic liability between personality and psychiatric disorders.

BRAIN CHANGES IN PSYCHOSIS AND DEPRESSION

Patients developing schizophrenia demonstrated decreased prefrontal cortical perfusion at their first presentation, compared with patients having a briefer schizophreniform illness. Molina *et al* (pp. 203–208) followed-up first-onset psychosis patients over a 2-year period, divided them into those developing schizophrenia or a brief psychosis and examined their PET images during an attentional task. They suggest that this hypofrontality may be an early biological marker of schizophrenia. Frontal cortical hypofunction was also evident when patients with schizophrenia, with established disease, were examined using fMRI while they produced spontaneous speech in response to Rorschach inkblots. This appears to be a real merging of biological and dynamic approaches! Kircher *et al* (pp. 209–214) suggest that this failure of frontal, and related temporal cortical, activation may contribute to patients' impairment in producing grammatically complex sentences. Hickie *et al* (pp. 197–202) used structural MRI to demonstrate that significant hippocampal volume deficits were present in individuals with major depression and that these deficits were more pronounced in older patients, those with late-onset disorders and those with melancholia. They also correlated with general cognitive and memory decrements, although volume reductions were unrelated to apolipoprotein E status.

AGGRESSION, SELF-HARM AND SUICIDE

Managing aggression and potentially aggressive people is an important component of forensic psychiatric care. Cure *et al* (pp. 185–189) carried out a review of clinical trials relevant to aggressive behaviour or people and found the data widely distributed across a range of different databases, with no single database providing a more definitive source than the others. They found multiple interventions per study, with wide variations in the quality of outcome measures and reporting. There were new scales used in many of the reports, with a high proportion of these being unvalidated. The authors suggest that wider collaboration, rationalising treatments and simplifying outcome measures might be a useful approach for similar studies in the future. Carter *et al* (pp. 253–257) demonstrated that increased severity of attempted self-poisoning was associated with a higher risk of subsequent completed suicide. The risk was greatest in those with serious physical harm, low coma scores, and increased severity of poison exposure. The authors suggest that such high-risk individuals may be suitable for more intensive attention after their self-poisoning attempt.

RISK OF RECURRENCE AFTER PUERPERAL PSYCHOSIS

Robertson *et al* (pp. 258–259) followed-up women presenting with a bipolar affective puerperal psychosis over a 9-year period. They found that 57% of these women experienced an additional puerperal psychotic episode, and 62% suffered a non-puerperal affective relapse. They make the very useful points that avoiding further pregnancy is no guarantee of avoiding subsequent illness, and that a positive family history of mental illness was associated with earlier non-puerperal recurrence, and may be relevant if prophylaxis is being considered.

https://doi.org/10.1192/bjp.186.3.177-a9 Published online by Cambridge University Press