Highlights of this issue

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IMMIGRATION, SCHIZOPHRENIA AND SOCIAL ADVERSITY

The issue of immigration remains controversial for many diverse reasons. One area of interest to psychiatry is the relationship between migration and mental illness. This has particularly interested researchers with regard to the incidence of schizophrenia. In this issue, Cooper (pp. 361–363) reviews the evidence for the role of socio-economic status as a mediator of the association between the higher rates of schizophrenia reported in some immigrant groups. He suggests that the evidence supports a primary social rather than genetic cause and that childhood social disadvantage, or increased adversity, may play a pivotal role in the aetiology of schizophrenia – a proposition supported by Patino *et al* (pp. 442–443), who demonstrate higher rates of psychotic symptoms in children from first- or second-generation immigrant families. Psychotic symptoms were more likely to be present in migrants who also fulfilled criteria for having family dysfunction, suggesting that immigrant status and family dysfunction act in a synergistic manner. The authors suggest that psychosocial stress associated with family dysfunction may prove to be the mediator of this relationship.

THE KRAEPELINIAN DICHOTOMY REVISITED

The distinction made by Kraepelin between bipolar affective disorder and schizophrenia is reaching the end of its useful life. Well, this is the view of Craddock & Owen (pp. 364–366), who review evidence from both epidemiological and molecular genetics research to conclude that psychiatry will be better served by conceptualising a spectrum of functional psychoses. The interplay between the risk genes and environmental factors for each individual will determine the clinical phenotype or expression of illness. Still examining the traditional dichotomy, McDonald *et al* (pp. 369–377) used an automated analysis of whole-brain volumetric magnetic resonance imaging data to demonstrate cortical grey matter deficits in patients with schizophrenia, while a comparison group of patients with bipolar disorder showed no such differences. In support of the spectrum of psychosis, the same study demonstrated overlapping white matter deficits common to both groups of patients when compared with healthy subjects. McIntosh *et al* (pp. 378–385) examined neuropsychological data from patients with schizophrenia or bipolar disorder and their unaffected relatives and reported that intellectual deficits were associated with the genetic risk of schizophrenia, while memory difficulties were related to increased liability to both schizophrenia and bipolar affective disorder, and the impairment of psychomotor performance was related to the expression of psychosis in general. They did not find any neuropsychological impairment specific to bipolar disorder.

SCHIZOPHRENIA – PREMORBID SYMPTOMS AND OUTCOME

There has been much recent interest in the concept of prodromal features and the importance of the first-onset period in psychosis; two papers in this issue address these questions. Owens *et al* (pp. 386–393) used Present State Examination data from two different groups, one a prospectively identified group of well participants at high risk of developing schizophrenia and the other of first-onset patients with a diagnosis of schizophrenia, to show that there were high levels of affective symptoms in participants prior to the onset of their illness. They discuss the difficulty in differentiating the premorbid from the prodromal phase of the illness. Rosenbaum *et al* (pp. 394–399) describe the results of a 1-year follow-up of a cohort of patients with first-episode psychosis treated with different psychotherapeutic approaches, in addition to a patient group given standard care. All of the patients improved over the period of the year, with no significant differences evident in the groups offered additional psychotherapeutic interventions. However, there was evidence that those patients without significant alcohol or drug misuse showed an enhanced response to additional psychotherapy.

DEPRESSION – DETECTION, TREATMENT AND PREDICTION

Birth weight has been used as a proxy for poor intra-uterine growth and has been associated with increased risk of cardiovascular disease and, in some reports, with increased risk of depressive illness. Osler *et al* (pp. 400–403) examined this risk in a birth cohort of men born in Denmark and found no relationship between low birth weight and admission to hospital for depression. In an editorial O'Keane & Scott (pp. 367–368) agree that the health status of an infant at birth is a determinant of long-term health, but highlight the complexity of the multiple medical, obstetric and psychological events during pregnancy, which feed into a single, relatively crude, measure such as birth weight or the presence of obstetric complications. They emphasise the role of poor maternal mental health during the pregnancy and exposure to psychological trauma during childhood, which, via effects on the hypothalamic–pituitary–adrenal axis, may provide a mechanism for the subsequent development of mood disorder. On a more pragmatic note, Eisses *et al* (pp. 404–409) demonstrated that there may be benefits in training staff in residential care homes for the elderly to detect depressive illness in this vulnerable group; and Schulze-Rauschenbach *et al* (pp. 410–416) report that transcranial magnetic stimulation is as efficacious as electroconvulsive therapy in the treatment of refractory depression, but with more favourable effects on cognitive performance and memory.

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