

## Highlights of this issue

By Sukhwinder S. Shergill

### First-episode psychosis: anxiety, drugs and neurological signs

The fruits of the burgeoning interest in first-episode psychosis are evident in this issue of the *Journal*. Janssen *et al* (pp. 227–233) examined neurological soft signs in adolescents with first-episode psychosis and found that increased severity of these signs was associated with failure of sensory integration correlated with smaller volume of the thalamus. However, the severity of soft signs related to motor sequencing was associated with smaller right caudate volume. They suggest that if these findings are replicated, then they offer the opportunity for a simple clinically accessible assessment of regional brain changes. Drug misuse is common at the age when most first-episode psychosis becomes apparent. Turkington *et al* (pp. 242–248) examined the differences in outcome between individuals with first-episode psychosis who continue to misuse drugs and those who either never misused them or stopped at the onset of the psychosis. Perhaps unsurprisingly, continued drug misuse was associated with more severe depressive illness, poorer functional outcome and greater relapse at follow-up after 1 year. More hearteningly, there was no difference in outcome between those who stopped using drugs after the onset of their illness and those who never used them – the authors suggest that identification and treatment of substance misuse needs to form an integral part of the services offered to these patients. The role of anxiety in the genesis of the early phase of psychotic illness has become increasingly clear. Michail & Birchwood (pp. 234–241) examined the rate of social anxiety disorder in first-episode psychosis, and report that 25% of a group of 80 patients fulfilled ICD-10 diagnostic criteria for anxiety disorder, with a further 11% reporting difficulty in social interactions. The authors conclude that social anxiety is present early in the course of psychosis and becomes more clinically prominent after the onset of psychosis. They did not favour the view that social anxiety is a secondary consequence of psychotic symptoms, and emphasised the importance of future work to tease out the time frame of anxiety symptoms relative to psychotic symptoms – whether they pre-date the psychosis or co-occur.

### Depression: genes, outcome and side-effects

There has been an exponential increase in gene–environment research aimed at identifying the genetic and environmental mechanisms underlying illness. However, what are the implications for the individual? It is now possible to obtain one's genetic profile through the world wide web; Newson (pp. 189–190) questions the ethical implications of genetic testing, and uses the example of the 5-HTT gene and the risk of depression.

She highlights the difficult questions relating to whether counselling is necessary, and whether testing should be available presymptomatically for diagnostic or screening purposes or only once the illness has been identified. Should children be screened? If so, who should provide consent, and is it ethical to do this in children if there is no clear effective intervention? There may be no good answers but the questions need to be considered, as the genetic genie is emerging from the bottle and may not easily be put back. Uher and colleagues (pp. 202–210) examine side-effects in one large, pharmacogenetic study of antidepressant efficacy. They found good interrater reliability between self-report and psychiatrists' ratings of side-effects, but intriguingly many more side-effects were reported when unmedicated than when taking medication. Thus, dryness of mouth was more frequent during treatment with escitalopram, but palpitations and headaches were more frequent when on no medication. A head-to-head comparison of escitalopram and nortriptyline showed increased diarrhoea, yawning and insomnia with escitalopram and increased dry mouth, blurred vision, dizziness and weight gain with nortriptyline. The authors conclude that self-report measures may provide a convenient and useful means to assess side-effects of antidepressants, and the increased rates of adverse effects reported by unmedicated patients may reflect an association of increased side-effects with the severity of depression. Johnson and colleagues (pp. 264–265) investigated the consequences of having minor depressive disorder during adolescence, finding that this was associated with increased rates of major depressive disorder in adulthood, as well as increased rates of anxiety and disruptive disorders in early adulthood. Interestingly, some of these effects were less evident in mid-adulthood.

### Capacity, detention and longitudinal studies

The treatment of patients with mental disorder without their consent can now be done through two separate legal frameworks – the Mental Health Act 1983 or the Mental Capacity Act 2005. Owen *et al* (pp. 257–263) report on the overlap between these: 24% of in-patients were found to lack capacity, but were informally admitted to hospital; a smaller 6% were detained under the Mental Health Act but retained capacity. They discuss the issues raised at the interface between these two frameworks and how an understanding of objection to treatment and deprivation of liberty safeguards is becoming a necessity for all clinicians. Longitudinal studies have been the source of significant advances in psychiatric research; however, Wolke *et al* (pp. 249–256) show that participant drop-out from such studies is not random and in their sample, more severely affected participants were more likely to drop out of the study – causing an underestimation of the prevalence of risks. However, the regression models they used in analysis were only marginally affected by these issues – suggesting that aetiological models from such longitudinal studies are valid and robust under appropriate conditions.