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Highlights of this issue

By Sukhwinder S. Shergill

The problem with DSM-5; young people, depression and psychosis

The recent publication of the DSM-5 has raised the profile of classification systems for disorders of mental health. An editorial by Frances & Nardo (pp. 1–2) highlights several problematic areas within the new DSM-5, including an increase in sensitivity without a corresponding attention to specificity, problems with dimensional systems focused on personality disorder, and the porous nature of the definitions of milder 'major' depressive disorder and anxiety disorders. They make an argument for the new ICD-11 to be aware of the mistakes made in DSM-5, to set higher scientific standards and to ensure more dialogue with differing viewpoints. In a similar vein, Koukopoulos and colleagues (pp. 3–5) focus on the DSM-5 criteria for depressive disorder with mixed features. They use their editorial to raise their fear that this will lead to more misdiagnosis and less appropriate treatment, and suggest that mixed depression should have a separate identity, characterised by core psychomotor agitation. Depressive illness has often been associated with diabetes and cardiovascular disease, but whether this was secondary to the impact of depressive illness itself, or as a consequence of shared aetiological factors, was not clear. Mannie *et al* (pp. 18–23) assessed healthy young people with a family history of depressive illness, and found they were more likely to have higher blood pressure and reduced insulin sensitivity. They conclude that depression, diabetes and cardiovascular disease appear to share common pathophysiological mechanisms – but were able to exclude a significant role for inflammation or cortisol hypersecretion as the mediating factors. The prognosis of first-onset psychosis is influenced by the duration of untreated psychosis (DUP). Birchwood and colleagues (pp. 58–64) report that a third of people presenting to early intervention services in Birmingham had a DUP of over 6 months, which, paradoxically, increased once they entered the mental health system. Given that this was observed in patients with access to a long-standing and well-established early intervention service, the authors suggest that there are structural barriers in the current delivery of services that prevent early intervention services from effectively reducing this important DUP.

Status, dementia and anxiety disorders across the age spectrum

Several diseases are differentially influenced by socioeconomic inequalities. In cardiovascular disease and in dementia there is evidence that these social inequalities may play a role in the aetiology of the disorder. Russ *et al* (pp. 10–17) found an association between leaving full-time education at an early age and subsequent death from dementia – but only in women. They highlight the challenge to the prevailing view that greater cognitive reserve is protective against the development of dementia, which does not account for the observed gender difference. Anxiety disorders are common, and there are some data suggesting that they are more difficult to treat in older patients. Wetherell and colleagues (pp. 65–72) confirm this hypothesis in a large sample of patients with anxiety disorder, reporting that both medication and psychotherapy may be less effective in older individuals with anxiety. However, they are clear that older people do benefit from treatment, although it appears that those older patients with generalised anxiety disorder or panic disorder may require far more intensive treatment to achieve optimal outcomes. An accompanying editorial by Oude Voshaar (pp. 8–9) argues that there is a two-fold problem: first, the generally low rates of treatment of anxiety disorder in old age need to be addressed; and second, there needs to be more attention given to developing age-specific treatment strategies.

Mental health, ethnicity and cognition in cocaine misuse

Mental health problems are evident in between 10 and 13% of children and adolescents in the UK, with similar rates in other countries and in different cultures. However, there is a trend from UK general population surveys that children of Indian origin have lower rates of mental illness than White British children. Dogra and colleagues (pp. 44–50) selected their sample of adolescent children to address this question. They report that adolescents of Indian ethnic origin did indeed have better mental health than their White counterparts. They review possible causes, including the effects of differences in parenting and social support networks, and potential beneficial effects of cultural identification and minority status when living in areas with higher concentrations of immigrants. Cocaine is the second most widely used illicit drug in Europe, and is commonly viewed as being relatively benign, often portrayed in the media as part of a Champagne lifestyle choice. However, contrary to this view, Vonmoos *et al* (pp. 35–43) found that dependent cocaine use was associated with significant cognitive impairments across a range of domains. Interestingly, recreational use of cocaine was also associated with significant deficits in some cognitive domains, particularly attention and working memory. The authors found earlier age at onset of use was associated with greater detrimental effects, and suggest that the observed deficits are underpinned by cocaine-induced changes in prefrontal cortical function.

Tardive dyskinesia

The biological basis of the involuntary movements that characterise tardive dyskinesia remains obscure. Tardive dyskinesia is present in some patients with untreated schizophrenia, but also appears to be related to antipsychotic treatment. Sarró *et al* (pp. 51–57) use brain imaging to demonstrate a reduction of brain volume in some key subcortical structures associated with tardive dyskinesia; the caudate nucleus and putamen were affected while the globus pallidus was spared. The authors are suitably cautious in interpreting their findings – the cross-sectional design precludes a clear cause and effect. Although antipsychotic treatment might cause a decrease in these brain volumes and thus lead to the development of tardive dyskinesia, it is equally possible that patients with existing smaller brain volumes in these areas may be differentially at higher risk of developing tardive dyskinesia. An accompanying editoral by Liddle (pp. 6–7) reviews the data from similar brain imaging approaches applied to other symptoms in schizophrenia, and also the effects of antipsychotic treatment on brain structure. He cautions against a simple attributional link between changes in brain structure due to antipsychotic medication and the development of symptoms, and highlights the role of ageing in the development of tardive dyskinesia, to illustrate the multifactorial contributors to its pathophysiology.