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Highlights of this issue

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Breaking the mould

When you cast your mind back the core phenomenology of psychosis, what do you remember? You will probably not have forgotten that psychological approaches often play second fiddle to pharmacological treatments. Concern about relapse through reflection on abnormal beliefs or experiences has meant that such psychological interventions are used sparingly. Or often not at all. It is refreshing to see this addressed head on in an editorial by Böge et al (pp. 71–72). The delivery of mindfulness-based therapies by trained professionals that consider group size, duration of meditation, minimisation of silence and aspects of language, bodes well for its more widespread use in everyday clinical practice. If we do not embrace such therapies, we risk perpetuating stigma and reinforcing 'othering'.

Another editorial by Cuthbertson et al (pp. 73–74) is quite literarily ground-breaking. It is part of our phylogeny to have a unique connectivity with the natural world, but often problematic to take this into the realm of experimental paradigm where green variables are complex and difficult to disentangle. Yet, studies that have embarked on this journey show not only economic gains in improving well-being, but also outcomes from nature-based therapies that are similar to cognitive–behavioural therapy. A walk on the wild side is indeed a breath of fresh air.

Smoke without mirrors

Although tobacco smoking has reduced at a population level over the past few decades, it remains considerably higher in people living with severe mental illness. In a short report, Gilbody et al (pp. 95–97) introduces us to the Smoking Cessation Intervention for People with Severe Mental Ill Health (SCIMITAR+) trial. It drew upon pilot study data to design a randomised controlled trial to demonstrate the clinical effectiveness of bespoke smoking cessation programmes at 6 months, compared with usual care. In order to overcome a false positive result from lack of sufficient power to detect differences between groups after a longer follow-up, the authors incorporated pre-trial data. Quit rates were maintained at 12 months in the first study of its kind.

It would be a rum do in today's world of science not to explore both nature and nurture. With increasingly sophisticated methodologies to examine the association between risk factors and mental disorders, the use of Mendelian randomisation has the advantage of overcoming limitations such as bias, confounding and reverse causality. Two elegant studies examined the association between tobacco smoking and severe mental illness. Vermeulen et al (pp. 88–94) found that lifetime smoking or smoking initiation increased the likelihood of developing bipolar disorder, but there was no clear evidence for an increased or decreased risk of smoking heaviness in people living with bipolar disorder. Another sound piece of evidence for ongoing investment in smoking cessation programmes. But the same did not hold up for an association between smoking behaviours (smoking initiation, smoking cessation, age at smoking initiation, quantity of smoking) and schizophrenia in a study using single nucleotide polymorphisms (SNPs) by Chen et al (pp. 98–103).

Altered states

The study of brain anatomy and physiology has been ongoing for several decades, but genetics has added another addition to the research toolbox. A study by Caseras et al (pp. 104–111) explored the influence of deletions, insertions and duplications in DNA – also called copy number variants (CNVs) – of rare genes on brain structure in healthy individuals. This method overcame the impact of other confounders such as substance use, which would be more likely in people with this mental disorder. Although their findings supported an association between the presence of CNVs and reduced brain surface area, there was also an association with increased cortical thickness, as opposed to the expected finding of cortical thinning. It may be that other factors are involved that are unrelated to the genes examined in this study

To end our journey for this issue, we revisit SNPs and the risk of severe mental illness. This time, in their relationship with bipolar affective disorder, a mental disorder known to have high heritability. One such polymorphism is in the *CACNA1C* gene, which is known to influence calcium entry into cells, which, in turn, regulates the development of brain cells through brain derived neurotrophic factor (BDNF). Smedler et al (pp. 77–79) found that people living with bipolar affective disorder who carried one of the high-risk alleles in the *CACNA1C* gene had a lower ratio of BDNF to BDNF precursor and lower levels of BDNF in their blood and cerebrospinal fluid compared with controls. The field of genetics is clearly becoming not only more diverse but also far reaching in what it can tell us about brain structure and function.

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