Novel interventions in affective disorders

A New Year, but an old problem: our repertoire of evidence-based interventions in affective disorders needs expanding. Cognitive remediation therapy (CRT) is showing early promise, but to date most work has been in schizophrenia. Bonnin et al (pp. 87–93) randomised individuals with bipolar affective disorder to 21 weeks of functional remediation (a variation on CRT), psychodrama, or treatment as usual and showed that remediation produced a significantly greater improvement in psychosis functioning that persisted a year later. As with psychoses, functional outcomes in bipolar affective disorder are linked to neurocognitive performance, and traditional treatments do not tap into this. More work is required unpicking which aspects of, and individuals in whom, CRT is most effective. Disappointing data however – at least in terms of seeking new treatments – from Yatham et al (pp. 78–86), who found no benefit from adjunctive agomelatine use in acute bipolar depression. The results are especially sobering as we know that depressive episodes constitute the majority of illness burden in bipolar affective disorder but that standard depression pharmacological strategies do not apply.

Acceptance and commitment therapy (ACT) is a form of cognitive–behavioural therapy (CBT) that promotes effective personal acceptance despite difficulties – living to the full. Pots and colleagues (pp. 69–77) present the first randomised controlled trial of web-based ACT, comparing a nine-module online intervention with both a waiting-list control and expressive writing. Significantly greater short-term reductions in depressive symptoms were found following ACT, although expressive writing had similar results towards the 1-year follow-up point. The practical implementation and cost considerations are encouraging: at the beginning of 2016 we don’t need reminding that Santa didn’t bring us more therapists or shorter waiting lists.

Longer-term outcomes and risks

Mental ill health is frequently a long-term issue, but problematically much research is based upon shorter-term outcomes. Using data from The Netherlands Study of Depression and Anxiety (NESDA), Lamers et al (pp. 62–68) followed over 600 individuals with major depressive disorder (MDD) over 6 years. Perhaps unsurprisingly, those with a moderate subtype fared best, and baseline severity of symptoms was the best predictor of trajectory: their prevalence of MDD varied from 25 to 35% during the various follow-up periods, whereas the figures ranged from 39 to 60% for the more severe types. Among individuals in the more severe cohorts, trajectories were similar in most aspects, but suicidal thoughts and anxiety persisted longer for those in a ‘melancholic’ subgroup, whereas an ‘atypical’ cohort had the highest body mass index and prevalence of metabolic syndrome. So, assimilating into clinical practice, does delineating depressive subtypes help? For symptom severity, yes; for atypicality, the main finding would appear to be a longer-term physical health risk, although the authors argue that the increased suicidal thinking in the melancholic cohort carries a considerable burden.

Maltreatment is linked with subsequent antisocial behaviour, and some work has shown this to be magnified by a specific allele of the monoamine oxidase A gene (MAOA). Ouellet-Morin et al (pp. 42–48) investigated whether the nature of violence exposure is a factor in this relationship, evaluating retrospective histories and conducting semi-structured interviews with over 300 young male participants of the Quebec Longitudinal Study of Kindergarten Children. Non-linear relationships between the gene and violence were detected: the authors argue that genetic moderation might not be present across the full spectrum of violence, but emerges once a specific degree of violence is experienced, and varies according to different antisocial outcomes. In a systematic review and meta-analysis Fazel and colleagues (pp. 17–25) ask what happens following discharge from secure psychiatric hospitals. Their data encompassed over 12 000 patients, of whom 52% were violent offenders. Redmission rates have been lower in more recent studies, and indeed were less than among some comparative groups of prisoners. However, secure psychiatric units are more likely to take individuals with a smaller range of serious violent and sexual offences – where reoffending is less likely than with acquittal crime more typically seen in prisons – and also may select those predicted more likely to respond to care. The risks to the individual are perhaps too seldom considered in this population; these results demonstrated an all-cause mortality and suicide death rate similar to those seen in psychoses populations, suggesting these are illness- rather than institution-driven. The debates about secure hospitals have been multifaceted: in economically straitened times they grow in number while other in-patient services shrink, they get caught in stigmatising media campaigns about mental health ‘stranger danger’, and they have been argued to abet an insidious psychiatric reinstitutionalisation. The authors’ synthesis of the current literature supports their provision of therapeutic input: in the context of such units taking about a fifth of the total mental health budget, a challenging question is, at what cost do they attain these gains?

The eyes have it?

As we sit in the depth of winter, the psychology of seasonal affective disorder (SAD) will have an intuitive feel for many. One might predict that individuals with visual impairment would be less affected, but in a fascinating piece, Madsen et al (pp. 56–61) show us that the opposite is true after testing variation in mood among 1647 members of the Danish Association of the Blind and 2271 control participants. Those with visual impairment had significantly higher scores, and there were greater rates in women and younger individuals in both groups. Subanalysis of the 251 respondents who had no conscious perception of light at all showed them to have an intermediate degree of seasonality between the visually impaired and those with normal vision. Individuals with visual impairment and blindness can still have a fully functional non-image-forming visual system, for example through photosensitive retinal ganglion cells that, through the photopigment melanopsin, project changes in ambient light conditions to the brain. These findings support retinal dysfunction as a critical driver of SAD, although of course other genetic and psychological factors clearly also play a role. As yet, no good data exist on the effects of light therapy in visually impaired populations; such work is clearly now needed.

Finally, on behalf of the readership of the BJPsych, enormous gratitude and thanks to Sukhi Shergill for a decade at the helm writing the Highlights: as Sukhi himself would say, respect.