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The British Journal of Psychiatry

Transcranial direct current stimulation for acute major depressive episodes: meta-analysis of individual patient data
André Brunoni *et al*

Association between stressful life events and psychotic experiences in adolescence: evidence for gene–environment correlations
Sania Shakoor *et al*

Negative symptoms and longitudinal grey matter tissue loss in adolescents at risk of psychosis: preliminary findings from a 6-year follow-up study
Andrew McKechanie *et al*

White matter tract integrity in treatment-resistant gambling disorder
Samuel Chamberlain *et al*

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Cover picture

That was then, this is now (2015).
Graham Foster (b.1970)

Using Bermudian iconography, this painting (acrylic on wood 48" × 36") is a surreal interpretation of how we are affected by our memories, the good alongside the bad ones, and how they affect us as time passes. Some seem to linger, while others fade. I've painted a sculptural representation of these memories contained inside the head, the saw blades representing the negative ones, while the rainbow wheel within represents the positive ones. The rust and aging of the metal depicts the gradual fading of these negative memories over time, allowing the positive ones to chart the future.

Website: www.grahamfoster.com
Facebook: Graham Foster art
Instagram: Graham Foster art

We are always looking for interesting and visually appealing images for the cover of the *Journal* and would welcome suggestions or pictures, which should be sent to Dr Allan Beveridge, British Journal of Psychiatry, 21 Prescot Street, London E1 8BB, UK or bjp@rcpsych.ac.uk.

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Highlights of this issue

By Kimberlie Dean

Potential biomarkers in ADHD, bipolar disorder, autism and Huntington's disease

Cheung *et al* (pp. 548–555) have explored the cognitive and neurophysiological processes associated with persistence or remission of attention-deficit hyperactivity disorder (ADHD) during adolescence and early adulthood. The authors found that ADHD remitters differed from persisters on preparation-vigilance measures, IQ and actigraph count, but not on a range of other measures. They discuss the implications of these findings for the development of non-pharmacological interventions for ADHD such as cognitive training and neurofeedback. In pursuit of cognitive endophenotypes for bipolar disorder, Georgiades *et al* (pp. 539–547) employed structural equation modelling techniques using data from a sample of twin and sibling pairs. Delayed verbal recall/recognition and spatial working memory were correlated with bipolar disorder using a parsimonious AE model, at least before adjustment for affective symptoms, whereas IQ and visual-spatial learning using an ACE model remained significantly correlated after such adjustment. The authors comment on the limited likelihood of a single cognitive phenotype providing the genetic signature for bipolar disorder and thus the need to pursue models with multiple cognitive and other markers.

In examining the neural mechanisms underlying mentalising processes in those with and without autism spectrum disorder, Rosenblau *et al* (pp. 556–564) tested a new video-based functional magnetic resonance imaging (fMRI) task to better replicate real-life social interactions and found evidence to support a central role for the amygdala in relation to mentalising in both groups. The authors comment on the possibility that amygdala functioning might represent a biomarker for a range of mental disorders characterised by impairments in social cognition, including schizophrenia and borderline personality disorder. Developing effective interventions to modify disease course in Huntington's disease relies on the availability of biomarkers that can sensitively reflect decline across disease stages. Domínguez *et al* (pp. 571–578) utilised multimodal MRI in a longitudinal study and found that caudate neurodegeneration, particularly atrophy, appeared to be the best potential candidate for such a biomarker. Importantly, caudate volume was sensitive to neurodegeneration both before and after symptom onset and was associated with both clinical and disease severity.

Adolescents at risk of psychosis

Two papers in the *BJPsych* this month focus on psychosis risk during adolescence, one addressing development of negative symptoms and the other gene–environment correlations. Using tensor-based structural imaging techniques over a 6-year follow-up period in a sample of adolescents at risk of psychosis for cognitive reasons, McKechanie *et al* (pp. 565–570) found that development of negative symptoms was associated with grey matter loss in regions relevant to social cognition. The authors call for further research focused on the pathophysiology of negative symptoms in order to address the gap in evidence to support development of effective treatments. Focusing on the impact of stressful life events (SLEs) and psychotic experiences in adolescence in a twin sample, Shakoor *et al* (pp. 532–538) found that SLEs were correlated with positive psychotic experiences and that shared genetic influences explained much of the covariation between dependent SLEs (those reliant on the individual's own behaviour) and paranoia and cognitive disorganisation. With regard to the covariation between hallucinations, grandiosity and SLEs which was also found, both genetic and common environmental factors were important. The authors comment on the need for researchers to view certain environmental risk factors in the context of genetics and to avoid categorical views of such risk factors as being either environmental or genetic.

White matter integrity – in gambling disorder and major depression

Tentative evidence for abnormalities in white matter integrity in gambling disorder has previously been reported and when Chamberlain *et al* (pp. 579–584) tested a sample of participants with treatment-resistant gambling disorder, evidence of reduced fractional anisotropy in the corpus callosum and superior longitudinal fasciculus was found, in comparison with a control sample. Further evidence of white matter abnormalities correlating with disease severity was also identified elsewhere in the brain. The authors call for more research on larger samples using a range of imaging markers and including samples with unaffected first-degree relatives.

Abnormalities in white matter integrity and hypothalamic–pituitary–adrenal (HPA) axis functioning have both been implicated in major depressive disorder, but their interrelationship has not been examined. In a diffusion tensor imaging study using tract-based spatial statistics, Liu *et al* (pp. 585–590) found evidence for an association when a sample of drug-naive patients with a first episode of depression were compared with controls – fractional anisotropy values in a number of regions were negatively correlated with serum cortisol levels in the case group. The authors propose that high cortisol levels may result in injury to microstructures in specific white matter circuits.