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Highlights of this issue

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It cannot be bargained with, it cannot be reasoned with…it does not feel pity or remorse or fear…and it absolutely will not stop. Ever

Attachment theory always reminds me of College membership MCQ exams: attachment theory = Bowlby; primal scream therapy = Janov; and then those odd questions about baboon social behaviour (whose hobby horse was that?). I came to re-appreciate attachment theory in a more matured way later on working with individuals with personality disorder. Writing in this month's *BJPsych*, Gwen Adshead advocates (pp. 511–513) that it is a critical biobehavioural stress management system about which psychiatrists need to be more *au fait*, including consideration of how their own attachment systems are activated by distressed patients. Indeed, 40% of the general population may have insecure attachments, a figure astonishingly believed to be doubled in clinical cohorts, and it may be regarded as a mediating factor when exposed to other stresses. There are growing data on the neurophysiology (and pathology) of attachment theory, and – in a more detailed way than contained in my fuzzy MCQ memories – Adshead recommends that its emerging neuroscience should be included in the ongoing trainee syllabus redesign.

Tiffin & Paton discuss (pp. 509–510) something that could never have been included in College exams when I took them – machine learning. The phrase sometimes feels a bit of a nebulously and suspiciously soft catch-all answer, like 'big data', 'genome-wide association study' and 'soft-Brexit'. Perhaps I am being cynical: let us look at the specific examples put forward in this editorial. Electronic patient records, including free-text entries, are seen as an obvious target, analysing natural language inputs to interpret symptom profiles and produce diagnoses. A follow on is to merge clinical changes with psychosocial issues, demographics and – putatively – biomarkers (although see later in this column) to produced stratified or personalised medicine for clinical subgroups, and prospectively predict outcomes. I enjoyed the authors' allusion to *The Terminator* film in the piece; will psychiatrists be replaced by DSM cyborgs in the future?

Hasta la vista, baby

We are beginning to unpick subtypes and their outcomes in attention-deficit hyperactivity disorder (ADHD). Some months ago, Kaleidoscope reported that a large majority of 'adult-onset' cases were actually likely to be primarily substance use disorders. This month Agnew-Blais *et al* (pp. 526–534) explore longitudinal outcomes in a child and adolescent cohort, following them up from ages 5 to 18, and delineating three clinical patterns: remitted, persistent and later onset. One might speculate, for example, that it is early disruption to education that causes enduring, irrecoverable damage even if ADHD abates with time; conversely might it be current symptoms that have the most impact on functioning? In this piece all groups had worse outcomes when compared with healthy peer-comparators, but there were differences between the subgroups. Remitted ADHD still led to socioeconomic and physical health deficits, fitting with the 'early disruption' model; however, the problem was particularly notable in the persistent and later-onset groups, with additional mental health, substance misuse and psychosocial problems. The potential confounders of childhood IQ, conduct disorder and other shared familial traits did not have an impact on the results. It is clear that those with ADHD often have poor outcomes; this work shows that it varies with the trajectory of symptoms.

Meier *et al* follow the line of thought, asking (pp. 555–560) if ADHD and anxiety disorders in childhood might be associated with the subsequent development of bipolar affective disorders. They utilised a Danish cohort study of almost 2.5 million individuals, following them up from their 16 birthday for at least 18 years. Having either ADHD or an anxiety disorder significantly increased the risk of developing bipolar affective disorders – by relatively similar amounts, about 10-fold – but having both led to a *30-fold* increase in incidence compared with individuals without these conditions. Dysregulation of internalising and externalising, from anxiety and ADHD, respectively, are argued to be a putative index of early manifestations of a liability to develop bipolar disorders.

Underneath it is a hyperalloy combat chassis, microprocessor-controlled, fully armoured. But outside, it is living human tissue

More on bipolar affective disorder in this month's *BJPsych*; specifically how to detect or stratify risk for it, from genetics through neuroendocrine functioning to neuroimaging. Calafato *et al* (pp. 535–541) note the increasing background evidence for a shared genetic propensity with schizophrenia; although individual gene variants might only convey small risks, at a polygenic level this might be more significant. In their genome-wide association study they found that those with psychoses had very significantly higher polygenic risk scores than healthy controls. The kicker is at the end; the accuracy of the predictive models was limited, and the authors rightfully note that the findings are not yet ready for translation into clinical practice. The model's area under the curve was 0.7 for schizophrenia and 0.65 for bipolar affective disorders – figures of >0.9 are considered to have high predictive discriminatory power.

Rowland *et al* (pp. 514–525) pleasingly resist the recent trend to force the word 'hot' into the title of a piece on inflammatory markers; a development surpassed more generally only by papers including the phrases 'mind the gap' and 'lost in translation' – we all hate the cheap trick of trying to enliven dull copy with easy cultural references. In this piece they meta-analysed inflammatory markers, neurotrophins and oxidative stress markers in individuals with bipolar disorders compared with controls, which included data from nearly 5000 participants. Fourteen relevant factors were identified from the 53 studies, although no one marker of itself could differentiate the mood phase of the condition. However, there was an argument that the combination of C-reactive protein/interleukin-6, brain derived neurotrophic factor/tumour necrosis factor (TNF)-α and soluble TNF-α receptor 1 having diagnostic value. For the clinicians among us, genome-wide association studies and neuroinflammatory markers sometimes feel forever tantalisingly just out of practical reach. What about neuroimaging? Using fractional anisotropy Foley *et al* (pp. 548–554) show significant differences in the uncinated fasciculus and cingulum that could delineate those with bipolar disorder type I from those with type II as well as compared with unaffected siblings and healthy controls.

Finally, Kaleidoscope (pp. 567–568) provides an answer to a question debated over the ages – who tells more lies, men or women? Place your bets and turn to the back to see if you are as correct as you smugly suspect you are.

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