Early intervention and pathways into care

Ghali et al. (pp. 277–283) contribute to the ever burgeoning literature on pathways to care and early intervention in psychosis by focusing on ethnic variations. In a naturalistic cohort study involving participants receiving care in eight London early intervention services, those in the White British group had the longest duration of untreated psychosis (DUP) and were more likely than their Black British counterparts to visit a general practitioner. All Black and minority ethnic groups were more likely to access emergency services and all Black patient groups were more likely than their White British counterparts to experience involvement with the criminal justice system. Importantly, the authors comment that the introduction of early intervention services has failed to have a substantial effect on DUP or on routes into treatment, across ethnic groups.

In an invited commentary, Warner (pp. 284–285) argues that early intervention services have been developed on the basis of a flawed model since mode of psychosis onset rather than DUP predicts outcome. In a linked editorial, Fearon (pp. 249–250) also acknowledges the link between DUP and mode of onset but cautions against abandoning pursuit of DUP reduction, and challenges early intervention services to consider the merits of broadening their remit beyond psychosis and forging better links with other agencies catering for the needs of young people.

Relevant to the discussion of DUP and mode of illness onset, another editorial in the Journal this month considers the value of a clinical staging model in psychiatry (Scott et al., pp. 243–245). The authors ask whether a staging model such as has been proposed for psychosis can be applied across psychiatric disorder or whether such models must be diagnosis-specific. Also of relevance to debates about pathways to care and service models, Thornicroft & Tansella (pp. 246–248) discuss the question of what balance to strike between hospital-based and community-based services in psychiatry. The authors conclude that evidence is lacking to justify an emphasis on one or other mode of service delivery and that a balance is required which will need to be locally informed and capable of responding to changing circumstances.

Antidepressant use in bipolar disorder

In the context of the controversy over the use of antidepressants in rapid-cycling bipolar disorder, Amsterdam et al. (pp. 301–306) utilised data from a randomised controlled trial of fluoxetine vs. lithium monotherapy for patients with bipolar II disorder initially stabilised on fluoxetine. Depressive relapse and treatment-emergent mood conversion rates were found to be similar for both treatment groups and not superior to placebo. No difference was seen for the rapid-cycling compared with non-rapid-cycling groups. In a linked editorial, Thase (pp. 251–252) comments that these findings potentially challenge the dogma relating to avoidance of antidepressant use in rapid-cycling bipolar disorder but reminds readers of the importance of considering an individual’s past response to different treatments. It may be that the only group for whom antidepressants should be avoided is defined by those who have previously experienced treatment-emergent affective shifts.

Biology of childhood maltreatment and trauma

McCrorry et al. (pp. 269–276) undertook a study of amygdala activation in a community sample of maltreated children and controls using fMRI and a paradigm involving pre-attentive presentation of angry, happy and neutral faces. They identified a heightened neural response to both positive and negative affect in maltreated children. The authors comment that given the fact that pre-attentive emotional processing is impaired in individuals with anxiety disorders, their finding in maltreated children may indicate a potential latent neural risk factor for future psychiatric disorder.

Consistent interactions between single nucleotide polymorphisms in the FK506 binding protein 5 (FKBP5) and childhood trauma have been found in relation to associations with stress-related mental disorders including post-traumatic stress disorder (PTSD) and affective disorders. Collip et al. (pp. 261–268) have extended such research to include models of psychosis and psychosis vulnerability in a range of samples. Their findings have led them to conclude that trauma may increase risk for psychosis via effects on the cortisol feedback loop similar to those for disorders such as PTSD, which supports the notion of underlying biological mechanisms crossing diagnostic boundaries.