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From the Editor's desk

By Peter Tyrer

The depressed chameleon

Have you ever seen a chameleon in its natural setting? I have, and the actuality is not nearly as exciting as the anticipation. Despite having an ability to transmogrify its appearance in a way that would excite every fashion designer, the chameleon does not glorify this asset, but sits passively, looking generally apathetic and, from my anthropomorphic viewpoint, seems sluggishly and conspicuously depressed. Perhaps this is apposite, as depression can appear in so many different forms. In this issue we see the chameleon of depression in at least four different colours: the deep, almost indelible, ultramarine of resistant depression (Fekadu *et al*, pp. 369–375); the chiaroscuro of bipolar depression (Tohen *et al*, pp. 376–382); the common or garden grey of standard depression (Kjaergaard *et al*, pp. 360–368); and the unusual scarlet of depression generated by cardiovascular disease (de Jongh & Roest, pp. 337–338; Åberg *et al*, pp. 352–359). The big question, asked over and over again in recent issues of the *Journal*[1–6] is, are these all part of the same disease or disorder, or do they represent different conditions? Goodyer (pp. 335–336) feels the answer can only come from mathematical, not clinical, models, and it is hard to defend our crude attempts to link diagnosis to treatment,[7] which have the same credibility as a drunk preaching the virtues of abstinence – the problem is obvious but the solution is not. If the depressive chameleon were the same irrespective of its colour, it would have a common core of clinical and biological features and a consistent natural history or, as Kendell puts it, it would have at least a reasonable degree of clinical validity.[8] What does seem to be clear is that depression, however classified, is not, and can never be, a unitary phenomenon. Like most other mental disorders it is also almost always associated with comorbidity but in our classification systems this is not adequately recognised.[9] Whether it is the depression or the comorbidity that makes the chameleon display in different colours is still uncertain, but when it comes to treatment the choice cannot be a common one. There is now abundant evidence that bipolar depression often does not respond to conventional antidepressant therapy and a range of other choices need to be considered,[10] including antipsychotic drugs, as Tohen *et al* (pp. 376–382) illustrate. In making the choice readers may be interested in the robust responses to our recent editorial[11] from a range of correspondents. Antipsychotic drugs are going to be an emotive subject for some time, if not indefinitely.

Camouflage is the aim of the chameleon, and one of the tasks of research papers is to identify the core of a subject that often appears obscured. Lawrence *et al* (pp. 344–351) and Orrell (pp. 342–343) cast more light on the voluminous but inchoate literature on psychological treatments for dementia. As Orrell comments, a few years ago psychological treatments would have been an afterthought in the therapy of this range of conditions as the bright glow of new and exciting drugs was spreading everywhere. The optimism has now dimmed a little, and several new compounds have not performed well in early studies, and we are having to take notice of an important range of non-pharmacological interventions that probably are effective but for which the evidence base remains relatively weak. I personally predict that these will turn out to be as least as good as drug treatment in improving the quality of life and satisfaction of patients with dementia as well as helping to solve other problems such as elder abuse,[12] and help to reduce the impact of yet another sombre colour of the depressive chameleon, the muddy brown of depression in dementia.

Griffith Edwards' legacy

Griffith (Griff) Edwards, formerly head of addiction services at the Institute of Psychiatry and the former editor of the journal *Addiction*, died on 13 September at the age of 83. My first training post was in psychiatry with Griff and I was immediately impressed with the fervour and enthusiasm of his teaching about a subject which I then had as much optimism about treating as training my cats to queue for their supper. But Griff's enthusiasm was infectious and before long we juniors were following in his footsteps, not always getting our footprints in the right place, but making sure we never forgot about the addictions whenever we assessed people in our clinics. We often grumble about psychiatric diagnosis but his paper with Milton Gross on the alcohol dependence syndrome[13] established the study of drug addiction firmly in the scientific arena and will never be forgotten. When Griff used to assess the stuttering formulations of diagnosis of both me and my fellow novice psychiatrist Derek Steinberg, he used to emphasise the need to distil these into 'pure gold'; he was the most notable exemplar of this advice.

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