

From the Editor's desk

By Peter Tyrer

Protecting the most vulnerable

We are used in psychiatry to dealing with problems that appear insoluble. One danger emanating from this place of woe is to develop a carapace of protective indifference. It therefore troubles me a little that recently we may have presented to our readers a surfeit of truths about child abuse as a precursor of mental illness.¹⁻⁴ If you hear an uncomfortable truth too often, it is forgotten, and I am worried that some of the truths about childhood maltreatment in all its forms might be dissociated by some into a distant memory store. But we have to pay attention to evidence when it grows stronger by the day. So in this issue we have a powerful combined message from Keyes *et al* (pp. 107–115) and Reid & Bentall (pp. 89–91), that even a small degree of prevention of childhood maltreatment would almost certainly have a big impact on the prevalence of not only common mental disorders but severe mental illness also. Yet despite this growing evidence we do not seem to be making much progress in preventing harm to this most vulnerable group in our community. As a psychiatrist who has seen adults with mental illness almost exclusively I have sometimes grumbled to my child psychiatry colleagues that they must have been selecting the wrong patients as so many with serious problems presenting in adulthood have not had their problems identified earlier. But I realise that the task of identification is far from easy, as so much childhood maltreatment is hidden, and even when exposed by those who have suffered, may not be believed (Read & Bentall, pp. 89–91). Problems in the governance of a family (or any other group in which children are gathered together) can be as difficult to expose as those in a complex organisation such as a bank. If things do go badly wrong, as they have done with so many banks in the past 5 years, all the covert activities of the organisation are exposed, but until they are laid bare in such a dramatic way, the internal system is powerful enough to keep intruders at bay. So it is with dysfunctional families and abused children; so many of these are able to present a demure and respectable face to the world until a tragedy, sometimes far distant from the original events, exposes the truth. So when I get frustrated at seeing the apparently irreparable damage done to an adult psyche and say to myself, 'Why couldn't this have been addressed by child psychiatric, psychological or social services in childhood, and then we wouldn't be in the mess we are in now', I need to be reminded of the ability of family systems to repel intruders and to cover up the truth. The great challenge of public health in the 21st century is to find a way of preventing abuse in all its forms, and as we become increasingly successful in doing this in the public arena and in promoting human rights across countries, family pathology flaunts itself even more prominently in our specialist services.

Barker *et al* (pp. 124–129) point to possible small ways in which prevention can be achieved, and Kirkbride *et al* (pp. 156–157), in exposing a higher than expected incidence of psychotic disorder in a predominantly rural setting, also pose the question, 'How many of these problems resolve without professional intervention and how much of the resolution is a consequence of innate resilience?'⁵ Choosing when to intervene still seems to be a lottery of doubt but increasingly, gene–environment interactions

(Fergusson *et al*, pp. 116–123)⁶⁻⁸ can not only point to those at risk but suggest new preventive strategies if we knew exactly when and where to focus them. We also need to be reminded that families are also a powerful source of strength and the consequences of sudden loss can be profound (Morina & Emmelkamp, pp. 158–159). Despite my gloom, I feel there are some glimmerings of light ahead; but Hilaire Belloc's advice to young boys, 'And always keep a-hold of Nurse, for fear of finding something worse', has now become considerably more complicated.

The bear necessities of depression

We still do not properly understand the origins of depression, its exact nature and function, and its common associations with early mortality (Markkula *et al*, pp. 143–149), and still seek earnestly confirmation that the treatments we hope are effective are indeed so (Leucht *et al*, pp. 97–106; Colloby *et al*, pp. 150–155). I have always been intrigued by the possible relationship between depression in humans and hibernation in mammals, especially in bears. Tsiouris⁹ proposed an interesting hypothesis that all the symptoms of mood disorder in man could only be understood fully by studying 'the entrance, maintenance, and exodus from hibernation in bears'. This might be considered a little far-fetched but both atypical forms of depression, characterised by fat storage through overeating, oversleeping and decreased mobility, and more typical forms, with withdrawal from the environment, lack of energy, loss of weight from not eating and burning stored fat, and changes in sleep pattern, when also associated with increased concentration of serum cortisol and a decrease in neurotransmitters such as noradrenaline, show that we are dealing with very similar phenomena. This hypothesis is also reinforced by the evidence of seasonal variation in depression with mid-winter being the most vulnerable time for post-partum mood disorders.¹⁰ Death is also frequent in hibernating bears¹¹ and so could explain the results of Scherrer *et al* (pp. 137–142) in their fascinating study. These findings are a challenge and an invitation, so we look forward to a new cohort of ursophile psychiatrists to elucidate, or at least expose, these bare essentials.

- 1 Kessler RC, McLaughlin KA, Green JG, Gruber MJ, Sampson NA, Zaslavsky AM, *et al*. Childhood adversities and adult psychopathology in the WHO World Mental Health Surveys. *Br J Psychiatry* 2010; **197**: 378–85.
- 2 Tyrer P. From the Editor's desk. *Br J Psychiatry* 2011; **199**: 172.
- 3 Bass C, Jones D. Psychopathology of perpetrators of fabricated or induced illness in children: case series. *Br J Psychiatry* 2011; **199**: 113–8.
- 4 Daly M. Childhood psychotic symptoms: link between non-consensual sex and later psychosis. *Br J Psychiatry* 2011; **199**: 251–2.
- 5 Rutter M. Resilience in the face of adversity: protective factors and resistance to psychiatric disorder. *Br J Psychiatry* 1985; **147**: 598–611.
- 6 Fergusson DM, Boden JM, Horwood LJ, Miller AL, Kennedy MA. MAOA, abuse exposure and antisocial behaviour: 30-year longitudinal study. *Br J Psychiatry* 2011; **198**: 457–63.
- 7 Alemany S, Arias B, Aguilera M, Villa H, Moya J, Ibáñez MI, *et al*. Childhood abuse, the BDNF-Val66Met polymorphism and adult psychotic-like experiences. *Br J Psychiatry* 2011; **199**: 38–42.
- 8 Artero S, Touchon J, Dupuy AM, Malafosse A, Ritchie K. War exposure, 5-HTTLPR genotype and lifetime risk of depression. *Br J Psychiatry* 2011; **199**: 43–8.
- 9 Tsiouris JA. Metabolic depression in hibernation and major depression: an explanatory theory and an animal model of depression. *Med Hypotheses* 2005; **65**: 829–40.
- 10 Sit D, Seltman H, Wisner KL. Seasonal effects on depression risk and suicidal symptoms in postpartum women. *Depress Anxiety* 2011; **28**: 400–5.
- 11 Stiner MC. Mortality analysis of Pleistocene bears and its paleoanthropological relevance. *J Hum Evol* 1998; **34**: 303–26.