

biological theories despite the preliminary evidence and go on to 'believe' the psychological theories without challenging the very basis of that belief.

Finally, in response to the issue of enhanced stigma associated with illness models, the study by Cunningham Owens *et al*<sup>4</sup> showing enhanced suicidality cannot be overgeneralised and it would be erroneous to undermine the well-recognised benefits and enhanced treatment adherence after psychoeducation. Patients have a 'right to know' about their mental illness. We can draw a parallel with HIV or cancer. Have we ever considered shifting away from their biological causation because of stigma or enhanced suicidal risk? How to educate and update the general public with the available information in the most appropriate way is the research question: concealing the evidence is unfortunately not an answer.

In contrast to the 1950s, thanks to the contribution from biological research, current clinical practice rests on a consensus that bipolar affective disorder, schizophrenia, obsessive-compulsive disorder and attention-deficit hyperactivity disorder are primary biological diseases with strong genetic components and psychosocial factors that contribute to the disease process. We agree with Young<sup>1</sup> when he brings up the bio-psychosocial model. Understanding all the complexities of biology is a 'process' and cannot be covered over a short period of biological research.

We can be optimistic at best and sceptical at worst about the clinical relevance of biological contributions but cynicism and dismissal would be a big mistake.

- 1 Kingdon D/Young AH. Research into putative biological mechanisms of mental disorders has been of no value to clinical psychiatry (debate). *Br J Psychiatry* 2007; **191**: 285–90.
- 2 Luborsky L, Rosenthal R, Diguera L, Andrusyna TP, Berman JS, Levitt JT, Seligman DA, Krause ED. The Dodo bird verdict is alive and well – mostly. *Clin Psychol Sci Pract* 2002; **9**: 2–12.
- 3 Messer SB, Wampold BE. Let's face facts: common factors are more potent than specific therapy ingredients. *Clin Psychol Sci Pract* 2002; **9**: 21–5.
- 4 Cunningham Owens DG, Carroll A, Fattah S, Clyde Z, Coffey I, Johnstone EC. A randomised, controlled trial of a brief interventional package for schizophrenic out-patients. *Acta Psychiatr Scand* 2001; **103**: 362–9.

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With Kingdon's view,<sup>1</sup> which seems to say that because we haven't found it we should not bother looking, all scientific endeavour would come to a halt. To propose that genetic research has not contributed to our ability to offer counselling is to ignore the extremely high heritability of bipolar disorder and the schizophrenias, and the advice we are able to offer in light of our knowledge. We have barely begun to skim the surface as far as research into the biological mechanisms underlying the major mental disorders is concerned, and more recent findings, such as the doubled or greater risk of developing a schizophrenic illness as a consequence of cannabis use, open yet more doors for researchers to explore the contents beyond. The fact that our tools are crude and our knowledge shallow does not justify giving up our search, as with this attitude no heavenly bodies, beyond those visible to the naked eye, would have been discovered. The biological basis of all the major mental illnesses, and their often successful chemical treatment, could only be dismissed by those blinded by dogma. The fact that our drug treatments have, for the most part, been discovered serendipitously does not render them any less valuable and to dismiss these discoveries would, for example, also

have led to the dismissal of the discovery of antibiotics or radiology. We have refined our treatments on the basis of many chance discoveries and long may the tradition of research for research's sake continue and thereby provide us with new therapeutic opportunities. The claims for cognitive therapy as the answer to all our problems are thankfully receding and allowing a more enlightened mindset to regain centre stage.

- 1 Kingdon D/Young AH. Research into putative biological mechanisms of mental disorders has been of no value to clinical psychiatry (debate). *Br J Psychiatry* 2007; **191**: 285–90.

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I would like to add briefly three further perspectives to the debate between David Kingdon and Alan Young,<sup>1</sup> on biological mechanisms and clinical psychiatry. First, it is unsustainable to contend, as Kingdon does, that biological approaches are based on the pursuit of physical causes for mental disorders. Causal processes in biology are both physical and intentional,<sup>2</sup> and modern biological psychology and psychiatry are making major contributions to our understanding of the interplay between them.

Second, as Young brings out, developmental studies show how social processes affect biology, and biology modifies susceptibility to environments. Animal studies find that early adverse experiences have long-term behavioural effects and an impact on biological processes such as gene expression.<sup>3</sup> Thus, links between quality of parenting in early life and subsequent adaptation may be mediated genetically.<sup>3</sup> Animal and human studies find that environmental effects on depression vary depending on genotype.<sup>4</sup> Studies of adult depression find that child maltreatment history modifies the role of interpersonal processes, the presence of structural differences in the brain, and treatment outcome, all highly relevant to clinical practice.<sup>5,6</sup> In studies of children, assessments of biological consequences of social experience, such as hypothalamic–pituitary–adrenocortical reactivity during parent–child conversations, are integral and essential. Developmental psychopathology would not have got off the ground based on the assumptions presented by Kingdon.

Finally, there is, in my view, a problem that is not to do with the conceptual and empirical issues debated by Kingdon & Young. Investigations of treatment outcomes, for example, in relation to genotype or maltreatment history, or genotype by maltreatment history, could be conducted within clinical practice but are very rare. As research funding, at least in the UK, becomes increasingly compartmentalised into different types of research such as 'health services', 'trials', 'basic sciences', who will fund the studies that cross these boundaries and bring biology into the clinic to the benefit of patients?

- 1 Kingdon D/Young AH. Research into putative biological mechanisms of mental disorders has been of no value to clinical psychiatry (debate). *Br J Psychiatry* 2007; **191**: 285–90.
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- 5 Teicher MH, Andersen SL, Polcari A, Anderson CM, Navalta CP. Developmental neurobiology of childhood stress and trauma. *Psychiatr Clin North Am* 2002; **25**: 397–426, vii–viii.

- 6 Nemeroff CB, Heim CM, Thase ME, Klein DN, Rush AJ, Schatzberg AF, Ninan PT, McCullough JP Jr, Weiss PM, Dunner DL, Rothbaum BO, Kornstein S, Keitner G, Keller MB. Differential responses to psychotherapy versus pharmacotherapy in patients with chronic forms of major depression and childhood trauma. *Proc Natl Acad Sci USA* 2003; **100**: 14293–6.

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**Author's reply:** Pattanayak & Pattanayak assert that I am dismissive of biological advancements. Not true (my hay fever benefits from them every summer). I'm simply trying to identify their relevance to psychiatric practice. If such advancements in understanding 'putative biological mechanisms of mental disorders' proved of concrete value to clinical psychiatry I would be delighted, but no single example has yet been given in these letters or the original debate which demonstrates this to be the case. Pattanayak & Pattanayak also say that genetic research helps in counselling patients but what they are describing is the relevance of discussing and providing evidence about the influence of family history. Any genetic component may relate to mental disorder but could it simply be the mediating effects of the genetics of vulnerability factors such as temperament?

Hill speculates on the interaction of biology and social circumstances and the former's possible role in conveying vulnerability to depression. Hypothalamic–pituitary–adrenocortical reactivity may be theoretically interesting but how does assessment of it contribute to clinical interactions? Also, how exactly,

in the management of childhood maltreatment are 'the presence [sic] of structural differences in the brain . . . highly relevant to clinical practice'?

If, after decades of watching the 'new dawn', the sun still does not appear to have risen, could I suggest to Dr Al-Adwani and his colleagues that they turn round and look in a different direction? They may find that it has been rising behind them, providing illumination of the way forward. Such a reorientation by psychiatry, to use psychosocial research as the foundation from which to address the major issues that exist, could transform our fundamentally flawed classification system,<sup>1</sup> appallingly stigmatising terminology<sup>2,3</sup> and manifestly confusing and ineffective explanations of mental conditions.<sup>4</sup>

1 McHugh PR. Striving for coherence: psychiatry's efforts over classification. *JAMA* 2005; **293**: 2526–8.

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3 Kingdon D. Down with schizophrenia. *New Scientist* 2007; **196**: 22.

4 Read J, Haslam N, Sayce L, Davies E. Prejudice and schizophrenia: a review of the 'mental illness is an illness like any other' approach. *Acta Psychiatr Scand* 2006; **114**: 303–18.

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