BJPsych | The British Journal of Psychiatry (2008) 192, 69–71.

Correspondence

Edited by Kiriakos Xenitidis and Colin Campbell

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Practical impact?

Tyrer, in 'From the Editor's Desk',[1] despite recognising the mundaneness of journal editors' preoccupation with impact factors, sings of the improved citation factor and high citation half-life of *The British Journal of Psychiatry*. While this is certainly praiseworthy and no doubt a result of the tireless efforts of Tyrer and a number of other people, it also raises the question of what the impact factor means to a clinician with a busy and well-habituated practice. The impact factor for them is an artificial statistic that may have no impact on their practice. It would be helpful to know whether there is a measure of the impact of a journal article on clinicians' practice and how journals perform on that measure. Citation statistics can be inflated by basic science or hypothesis-based or epidemiology-based articles (to name a few), and none of these may have any impact whatsoever on our day-to-day practice, whereas the much more lowly weighted case reports (remember Freud) can have a significant impact. Yet case reports may not be highly cited. If such a measure is indeed developed, the romantic song will then be even sweeter; and not at all mundane. Robert Burns would probably forgive then.

1 Tyrer P. From the Editor's Desk. *Br J Psychiatry* 2007; **191**: 188.

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doi: 10.1192/bjp.192.1.69

Author's reply: I share Dr Gangdev's concerns in part. The impact factor, an invention of Eugene Garfield,[1] is not a necessary part of science. It merely reflects our preoccupation with league tables in every part of life. Any senior professional, whether editor, headmaster, company director or hospital manager, likes to know exactly where their organisation stands with respect to others on at least an annual basis; this seems to be so much more important than non-numerical measures such as letters of appreciation or complaint. It therefore seems to have little relevance to readers of a learned journal, who are not the slightest bit interested in the level of inflation of the Editor's ego, but only in the content of papers published in the journal. There is now evidence that the impact factor does indeed provide a reasonable comparison of the relative quality of a journal; however, what it does not do, despite increasing claims to the contrary, is provide a valid 'assessment of the quality of individual papers, scientists and departments'.[2] All that can be said about the publication of a paper in a high-quality journal is that the review process is likely to have been carried out with a higher degree of precision and care than that for an equivalent paper in a journal of very low impact factor; therefore, in general, the reader can have more confidence in the presentation of the findings. This is not to say they are necessarily more accurate or of greater scientific significance, although in the broadest terms, they probably are.

But the highly informed reader can select good papers from poor ones without the aid of the impact factor, and the preoccupation of the scientific community with its importance sometimes approaches the ludicrous, such as with the research assessment exercise (RAE) in the UK, which demands articles from high-impact-factor journals, among other measures, in comparing the relative value of scientists. How a nutritionist or a historian can be validly compared with a psychiatrist is, in my view, intrinsically meaningless. I have helped colleagues who have decided to leave academia for a less topsy-turvy land with a set of verses, also derived in part from Robert Burns, and which include the following (sung to the tune of Auld Lang Syne as they make their last journey down the university corridor);

No longer will I troubled be
With targets to be won
Flush RAE down the lavatory
'Cos its impact factor's none.

Declaration of interest

P.T. is Editor of *The British Journal of Psychiatry*.

1 Garfield E. Citation analysis as a tool in journal evaluation. *Science* 1972; **178**: 471–9.

2 Opthof T. Sense and nonsense about the impact factor. *Cardiovasc Res* 1997; **33**: 1–7.

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doi: 10.1192/bjp.192.1.69a

Biology is psychiatry's new dawn

In his debate with Allan Young, David Kingdon[1] has provided his perspective suggesting the dismissal of biological advancements and the promotion of psychosocial research instead. This is our humble attempt to challenge some of the points raised by him.

To discard the biological advances for being unable to pinpoint the 'exact aetiology' or 'cure' is unjust. It has remained elusive in the whole of medicine (90% of hypertension is idiopathic sans any 'cure'; so is epilepsy). We never forget to take our antihypertensive pills – why make an exception for psychiatric illnesses?

As for the statement made by Kingdon, 'research into psychosocial mechanisms, which has been much more productive',[1] we refer to a recent meta-analyses by Luborsky.[2] These revealed that the effect size attributed to specific therapy techniques is only 0.2 and found common factors such as therapist–client alliance to be more important.[3] This casts doubts over the clinical relevance of 400 different types of psychotherapies. Absence of large-scale well-controlled trials on efficacy of psychotherapy *v.* pharmacotherapy in major mental illnesses further leaves us wondering. In addition, the abandonment of once prevalent theories about 'latent homosexuality', 'refrigerator mothers' and 'schizophrenogenic families' only begs us to be doubly cautious before accepting empirical evidence as absolute.

Those who don't learn from mistakes made in the past are condemned to repeat them. We quote this in the context of the past 100 years of dementia research. Alzheimer's initial findings were dismissed as non-specific and most tributes on his death in 1915 did not even mention his, now significant, discovery. Psychological theories of dementia ('elderly neglect/loneliness') were in vogue until the 1960s. Ironically, we often dismiss the

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*[4] 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[1] 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 contribtions 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, Diguer 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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doi: 10.1192/bjp.192.1.69b

With Kingdon's view,[1] 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 heritabilty 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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doi: 10.1192/bjp.192.1.70

I would like to add briefly three further perspectives to the debate between David Kingdon and Alan Young,[1] 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,[2] 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.[3] Thus, links between quality of parenting in early life and subsequent adaptation may be mediated genetically.[3] Animal and human studies find that environmental effects on depression vary depending on genotype.[4] 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.[5,6] 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.

2 Bolton D, Hill J. *Mind, Meaning and Mental Disorder*, (2nd edn). Oxford University Press, 2004.

3 Francis DD, Diorio J, Plotsky PM, Meaney MJ. Environmental enrichment reverses the effects of maternal separation on stress reactivity. *J Neurosci* 2002; **22**: 7840–3.

4 Caspi A, Sugden K, Moffitt TE, Taylor A, Craig IW, Harrington H, McClay J, Mill J, Martin J, Braithwaite A, Poulton R. Influence of life stress on depression: moderation by a polymorphism in the 5-HTT gene. *Science* 2003; **301**: 386–9.

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.

https://doi.org/10.1192/bjp.192.1.69b Published online by Cambridge University Press