monitor, document and report adverse events. Unfortunately, experience demonstrates that this is frequently lacking and can result in the delayed recognition of potentially serious side-effects and interactions.

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The antidepressant debate should move on

In her editorial Moncrieff (2002) ignored decades of work and focused on a few pieces of research, one of them from 1965. The editorial was followed by a letter criticising this view (Malt, 2002), which was, however, published under the title 'The antidepressant debate continues'. This title might leave the impression that the effectiveness of antidepressants is still questionable.

Some of our colleagues might conclude that antidepressants have no proven effect and their patients should discontinue them. The consequences of such actions have been researched extensively: the relapse rates are approximately twice as high for patients who stop their medication in the first 2–6 months beyond the point of remission, compared with those who continue treatment (e.g. Anderson *et al*, 2000; Hirschfeld, 2001). Other patients might be denied an effective treatment. Going through all the evidence, which includes comparisons with other treatments and between different classes of antidepressants, animal work, and tryptophan and noradrenalin depletion experiments in people responsive to antidepressants, would be like reinventing the wheel, and is not the subject of this letter. As the rest of us continue to learn of advancements being made to refine and improve the pharmacotherapy of depression, is it possible that there is a group believing that antidepressants really do not have an effect? There is indeed an antidepressant debate – but it is not whether they work but rather how they work that is the current focus of interest.

Declaration of interest

G.K. and A.K. have received grants from Janssen Research Foundation and GlaxoSmithKline for conducting molecular genetic studies in psychiatric disorders. G.K. has received honoraria from Janssen Cilag for delivering lectures.

Anderson, I. M., Nutt, D. J. & Deakin, J. F. W. (2000) Evidence-based guidelines for treating depressive disorders with antidepressants: a revision of the 1993 British Association for Psychopharmacology guidelines. *Journal of Psychopharmacology*, **14**, 3–20.

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In her editorial, 'The antidepressant debate', Moncrieff (2002) provocatively questioned the orthodox view that antidepressants are efficacious (i.e. work under clinical trial conditions) in the treatment of depressive illness. Questioning accepted views is valuable but Moncrieff missed the real question, which relates to effectiveness, that is when are antidepressants useful clinically? The efficacy argument at the head of her critique, based on individual, often old and poor-quality, studies flies in the face of consistent findings of antidepressant efficacy in systematic reviews and meta-analyses (e.g. Anderson *et al*, 2000). Even the argument of bias due to unblinding because of side-effects is contradicted by her own meta-analysis, which showed a significant benefit for antidepressants over 'active' placebo (Moncrieff *et al*, 1998). Even more compelling is the evidence from continuation/maintenance studies which show that antidepressants have a robust effect in reducing rates of relapse and recurrence (Carney *et al*, 2001), a cumulative effect over months or years. Explaining this by a placebo effect is difficult to accept, or else demands re-evaluation of the nature of placebo.

This is not to say that 'negative' studies, where antidepressants are no better than placebo, should be ignored. An important factor is probably related to severity of depression. Khan *et al* (2002) found that the proportion of studies favouring antidepressants over placebo increased with the severity of depression; the response to placebo declined with increasing severity whereas that to antidepressants increased. This raises the fundamental question of when (i.e. at what severity) in real life practice does someone with depression clearly benefit from antidepressant drug treatment. Put another way, is the current trend to wider use of antidepressants for milder depression justified? This can only be answered empirically in appropriate naturalistic trials, and even then will require value judgement about the size of the benefit.

Perhaps the most worrying aspect of Moncrieff's editorial was the implication that we should take either a psychosocial *or* a physical approach to the treatment of depression. Surely we should have put this rather tired dualist view of psychiatry behind us by now? A holistic view combining drug and psychological treatments is to be preferred and evidence is accumulating that this leads to better outcomes. To conclude, a balanced view of the evidence for antidepressants firmly places them as an established and important therapeutic option (alongside others) in the treatment of depression, with their role becoming more central with increasing severity. The true debate is about the best way to use them.

Declaration of interest

I.M.A. and P.M.H. have both received honoraria for speaking, been members of advisory boards, received research grants and had support for attending scientific meetings from several pharmaceutical companies involved in the manufacture and marketing of antidepressants.

Anderson, I. M., Nutt, D. J. & Deakin, J. F. W. (2000) Evidence-based guidelines for treating depressive disorders with antidepressants: a revision of the 1993 British Association for Psychopharmacology guidelines. *Journal of Psychopharmacology*, **14**, 3–20.

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Author's reply: I would like to make some comments on the points raised by Kirov & Korszun and Anderson & Haddad. They both cite evidence from continuation and maintenance studies, but this is likely to be more flawed than evidence from acute treatment studies. In studies of long-term treatment, patients who have responded to acute treatment are randomised to continue active drugs or to be withdrawn to an inert placebo. However, it cannot be assumed that the state of having had treatment withdrawn is equivalent to never having had treatment in the first place. It is known that there is a discontinuation reaction with all classes of antidepressants (Haddad *et al*, 1998). The symptoms of this reaction may themselves be mistaken for relapse, or they may unblind participants and predispose them to relapse because of fears of discontinuing treatment. This is likely to be a particular problem given that the initial sample of patients comprises people responsive to treatment who are therefore likely to have high expectations of the benefits of treatment.

In addition, the evidence on antidepressant effects and severity is complex. The majority of studies that show that increased efficacy correlates with increased severity are studies of out-patients. In in-patients, more-severe depression has been shown to respond less well to antidepressants than moderate depression does, independently of the presence of psychotic symptoms (Kocsis *et al*, 1990). In our meta-analysis we found no significant differences from placebo in in-patient studies (Moncrieff *et al*, 1998), which is in line with results from other large landmark in-patient studies such as the Medical Research Council study and the National Institute for Mental Health study described in my editorial (Moncrieff, 2002).

Finally, if the benefits of antidepressants are so obvious, it seems surprising to me that we have little evidence that the burden of depressive illness is reducing in line with the vast expansion in antidepressant prescribing. In contrast, long-term incapacity related to depression has been rising rapidly both in absolute terms and in relation to other conditions (Moncrieff & Pommerleau, 2000).

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Kocsis, J. H., Croughan, J. L., Katz, M. M., *et al* (1990) Response to treatment with antidepressants of patients with severe or moderate nonpsychotic depression and of patients with psychotic depression. *American Journal of Psychiatry*, **147**, 621–624.

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Talking about cognitive analytic therapy

Isaac Marks' review (Marks, 2003) encapsulates the reciprocal roles expressed in so much of the comparative debate in psychotherapy: dismissing : dismissed, contemptuous : contemptible. To contemptuously attack the review would simply be to continue the dance and to encourage further polarising responses. I have great respect for Isaac Marks' work and would invite him to join in a dialogue with cognitive analytic therapy. It was thought-provoking to consider the role of Pavlov in the developmental understanding of symptoms.

Cognitive analytic therapy has its devotees among therapists and clients. It is a tremendously human therapy where the strengths of cognitive theory and object relations theory have more recently begun to incorporate strikingly original ideas on human development, dialogue and the construction of interpersonal meaning from the Russian tradition. For many this represents an exciting evolution of thought concerning the nature of the psychotherapeutic relationship and the process of change in psychotherapy.

Cognitive analytic therapy has attempted to integrate the cognitive and the analytic as well as the dialogic Eastern approach to development with the reductionist Western scientific tradition. A more challenging task is to bring into dialogue the entrenched culs-de-sac of psychotherapy theory and their defenders. So, let's start to talk and engage in some positive role-play – valuing : valued, respecting : respected, giving : receiving.

Declaration of interest

J.H. is a member of the Association for Cognitive Analytic Therapy and has published in the field.

Marks, I. (2003) Book review: *Introducing Cognitive Analytic Therapy. Principles and Practice*, by A. Ryle & I. B. Kerr. *British Journal of Psychiatry*, **182**, 179–180.

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Preserve psychoanalysis from too much neuroscience

Professor Hobson (2003) argues admirably for the continued relevance of psychoanalysis in a mainstream psychiatric journal. But is his suggested rapprochement between psychoanalysis and contemporary neuroscience really desirable?

Contemporary neuroscience as illustrated by his example of 'mirror neurons' typically assumes an 'empiricist' worldview. In brief, imitation is assumed to be an acquired process in which information is abstracted from experience using associative learning. The current focus is on the anatomical location of the associative learning responsible for imitation (Rizzolatti *et al*, 2001).

In contrast, psychoanalysis derives from an older, rationalist philosophical tradition. It assumes the existence of both innate beliefs, such as persecutory anxiety, and distinct mental mechanisms, such as introjection or Klein's paranoid–schizoid position, that do not rely on associative learning.

These two philosophies have been in tension for centuries. One option is to make psychoanalysis more empiricist by downplaying the innateness and divergent mental mechanisms of classical theory. This is seen in attempts to incorporate 'theory of mind' deficits into a psychodynamic understanding of mental states (Fonagy, 1991).

But will associative learning form the secure basis for understanding the mind that empiricism proposed? Practical