should be told of the truth regarding statistically significant findings. These should be neither exaggerated nor minimised.

Finally, women who dare to express emotional trials following an abortion face rejection from people on both sides. A few pro-lifers harshly dismiss these women as 'sinners' who deserve a lifetime of grief. Conversely, at least a few pro-choicers dismiss their grief as 'whining' or 'rare', or suggest that only women mentally unstable prior to their abortions would complain so much. By contrast, the post-abortion healing movement simply asks those on both sides to respect the experiences of women grieving a past abortion. But even this pro-healing position is attacked. Pro-choicers accuse us of manipulating gullible women into falsely blaming unrelated life problems on their abortions.[6] Some pro-life advocates, meanwhile, accuse us of encouraging an unprincipled, narcissistic worldview that diminishes the moral absolutes regarding the sanctity of life.[5]

To my mind, the question of whether abortion is the sole, direct cause of certain mental illnesses is far less important than the fact that many self-aware women want help coping with a past abortion experience.[7] Why is it so hard to simply accept their self-assessments and stated needs? Women deserve better.

Declaration of interest

D. R. is Director of the Elliot Institute, USA.

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The fall of the aypticals?

In his editorial arguing that atypical antipsychotics can no longer be regarded as having advantages over conventional drugs, Kendall[1] makes two statements which do not do justice to the available evidence.

First, he states: 'With the exception of clozapine for treatment-resistant schizophrenia, the atypicals, as a group of antipsychotics, are no more efficacious for schizophrenia than the typicals, whether it is chronic or acute, for first or subsequent episodes, for the acute episode or for promoting recovery'. This is supported by a reference to the updated National Institute for health and Clinical Excellence (NICE) guideline for schizophrenia,[2] which in turn based its conclusions on a series of meta-analyses carried out by the National Collaborating Centre for Mental Health (NCCMH; www.nccmh.org.uk). The problem here is that two other meta-analyses have reached different conclusions. In 2003, Davis *et al*[3] found that, apart from clozapine, three atypicals showed significant superiority over conventional antipsychotics: risperidone (22 studies, effect size (ES) 0.25), olanzapine (14 studies, ES = 0.21) and amisulpride (12 studies, ES = 0.29). Six years later, Leucht *et al*[4] had closely similar findings for olanzapine (28 studies, ES = 0.28) and amisulpride (13 studies, ES = 0.31); the effect size had become smaller for risperidone, but it was still significant (34 studies, ES = 0.13).

One reason why the NICE/NCCMH meta-analysis may have reached negative conclusions concerning these three drugs is that it included fewer studies. The outcome measure used by Davis *et al*[3] and Leucht *et al*[4] was reduction in total symptom scores, based on pooled data from the Positive and Negative Syndrome Scale (PANSS), Brief Psychiatric Rating Scale (BPRS) and CGI (Clinical Global Impression) scale, and from the PANSS and BPRS respectively. In the NICE/NCCMH meta-analyses there were only 17 studies of risperidone, 10 of olanzapine and 4 of amisulpride in which the drug was compared with a conventional antipsychotic on any of these outcome measures. Data were also pooled separately for studies carried out on patients with first-episode schizophrenia, on those with acute exacerbations or recurrences, and on those with treatment-resistant illness. As a result, the maximum number of studies included in any of the NICE/NCCMH meta-analyses of overall symptoms for these three atypicals was six, and several contained only one or two studies.

Later in the article, Kendall cites approvingly a meta-analysis by Geddes *et al*[5] in 2000, which found evidence that the superiority of atypicals (including clozapine) was an artefact of the high dose of the typical antipsychotic used as a comparator in some of the trials. These authors used meta- regression to examine the predictive value of haloperidol dose (23 studies) or chlorpromazine dose (7 studies) on the outcome of total symptom scores. In both cases, the findings were significant: an observed advantage in favour of atypicals disappeared as the dose of the comparator drug decreased. Davis *et al*[3] subsequently explored the effect of comparator dose in their meta-analysis. The results of several different analyses led them to conclude that there was no significant effect of haloperidol in a larger data-set of studies. Leucht *et al*[6] also failed to find a significant effect of chlorpromazine comparator dose in another meta-analysis carried out at around the same time. Geddes *et al*[7] argued that a significant effect of comparator dose could be re-instated in this latter meta-analysis by using their own meta- regression technique; in their author reply, Leucht *et al*[7] countered that the effect was not significant when a variety of other statistical techniques were used, indicating that the finding was not robust.

Kendall states that the comparator drug effect has been 'neither confirmed nor disproved by later meta-analyses'. An arguably more accurate conclusion is that it was an early finding which has not stood the test of time.

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6 Leucht S, Wahlbeck K, Hamann J, Kissling W. New generation antipsychotics versus low-potency conventional antipsychotics: a systematic review and meta-analysis. *Lancet* 2003; **361**: 1581–9.

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Author's reply: Dr McKenna seems to have misread and misunderstood the editorial.[1] I do not argue that 'atypical antipsychotics' (whatever they are) can no longer be regarded as having advantages over 'conventional drugs' (whatever they are). I argue that the class – the 'atypical' antipsychotics – has been fabricated for marketing purposes and has no basis in science or clinical practice. Although some studies do suggest that individual drugs differ in terms of side-effects, potency, efficacy and effectiveness, the differences – with the exception of clozapine for treatment-resistant schizophrenia – are small, and their relative effects are, at least in part, dependent on the potency[2] and dose[3] of the comparator. These differences do not constitute a 'class effect'.

In the meta-analyses for the schizophrenia NICE guideline,[4] we examined the use of antipsychotics in a number of different clinical contexts (e.g. first episode, acute episode and treatment resistance) and concluded that the differences in efficacy between drugs were unlikely to be clinically important. However, the guideline did acknowledge, as do other meta-analyses,[3,5,6] that differences in terms of side-effects allow clinicians and service users to find a drug that suits them. Moreover, all three meta-analyses agree that there are no consistent differences or similarities between 'typicals' and 'atypicals'— this is an important perspective that McKenna seems to have missed.

In undertaking our meta-analyses for the development of a guideline, we were guided by a broad range of clinical review questions. The more specific the question the fewer studies are likely to be able to answer the question. The data underpinning the use of antipsychotics in the treatment of acute schizophrenia included over 72 000 patients, whereas for the first episode this figure dropped below 2000. We could have lumped more data together: it is very unlikely that increasing the numbers of studies and participants with different presentations in the meta-analyses would change the central conclusions (that oral antipsychotics are all much the same in terms of efficacy); but it would have significantly diminished the clinical utility of each analysis.

The study by Geddes *et al*[3] is important not only in highlighting the influence of the comparator dose on efficacy, but also in questioning the integrity and claimed superiority of the class of 'atypicals'. It is true that Davis *et al*[5] did not confirm the findings of Geddes *et al*;[3] nevertheless, I maintain that the findings have clinical face validity. Not irrelevant to this perspective is that Leucht *et al*,[2] in their paper summarising the debate, said 'It is a major limitation that only a few studies used mid-potency FGA [first-generation antipsychotic] comparators. We recommend that each new drug is compared with a low-potency, a mid-potency, and a high-potency FGA.' Explicit in this recommendation is that the potency of the comparator can introduce bias; it would be odd to suggest that the dose of the comparator would not also have an important effect. In any event, McKenna may be in danger of not seeing the wood for the trees: the 'atypicals' have surely fallen.

1 Kendall T. The rise and fall of the atypical antipsychotics. *Br J Psychiatry* 2011; **199**: 266–8.

2 Leucht S, Wahlbeck K, Hamann J, Kissling W. New generation antipsychotics versus low-potency conventional antipsychotics: a systematic review and meta-analysis. *Lancet* 2003; **361**: 1581–9.

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