Dr Cuijpers and Colleagues Reply

To the Editor: We thank Dr Gaudiano and colleagues for their contribution to the discussion about psychotherapy for dysthymia. We agree very much with Gaudiano et al that we should be careful about drawing definite conclusions about the comparative efficacy of psychotherapy on the basis of 5 trials. Therefore, we have been careful in our meta-analysis of comparative studies to describe this as an important limitation of our study, and we have repeatedly indicated that our results should be considered with caution and our conclusions should not be seen as definite.1

On the other hand, we do not agree with their interpretation of the evidence to date on psychological treatments of dysthymia. First, Gaudiano et al argue that 2 of the 5 trials were based on brief problem-solving therapy (PST)2,3 and that this treatment may be too brief for treating dysthymia adequately. This may be true, but as we described in our article, removal of these 2 studies did not reduce the effect size. On the contrary, as can be seen in Table 2 of our article, the effect size indicating the difference between psychotherapy and pharmacotherapy for dysthymia increased from 0.28 to 0.44 after removal of the PST studies. Further, as can be seen in Figure 1 of our article, in each of the 2 studies on PST, the
The difference between psychotherapy and pharmacotherapy was very small (nonsignificant effect sizes of 0.12 and 0.15). Apparently, PST does not do such a bad job compared to pharmacotherapy, even with only 4 to 6 sessions of 30 minutes!

Second, Gaudiano et al argue that interpersonal psychotherapy (IPT) may also be less effective in dysthymia because it was originally designed for acute depressive illness and may require further adaptation for chronically depressed patients. This could also very well be true and could give some explanation for our findings. However, the fact that IPT was not designed for chronic depression does not imply that it cannot be effective. Cognitive-behavioral therapy (CBT) and PST were also originally designed for acute depression, and above we saw that PST may be quite effective in dysthymia compared to pharmacotherapy.

However, we disagree most on the third issue Gaudiano et al bring forward. They say that the most appropriate interpretation of the evidence is that “comprehensive CBT adapted for chronic depression is a promising treatment for dysthymia.” In our meta-analysis, we included 1 study in which CBT was examined in patients with dysthymia. Of the 5 studies among dysthymia patients, this study showed the largest difference between psychotherapy and pharmacotherapy (effect size = 0.71 in favor of pharmacotherapy; see Figure 1 of our article). However, Gaudiano et al point to the larger retention rate in CBT compared with pharmacotherapy. And this observation is correct. In the pharmacotherapy condition, 6 of the 18 patients (33%) dropped out, while in the CBT condition only 3 of the 13 patients (23%) dropped out. It would in theory be possible that this difference in dropout rate (10%) is indeed in part responsible for the difference between psychotherapy and pharmacotherapy. However, it seems very unlikely that this difference can explain a differential effect size of 0.71.

Dr Gaudiano and colleagues bring forward that earlier research has suggested that psychotherapy for dysthymia may require a greater number of sessions than are typically used in acute depression treatment. However, this suggestion is based on an open study of 10 patients, which can hardly be considered a strong suggestion. Furthermore, we saw above that PST with only 4 to 6 sessions of 30 minutes is almost as good as pharmacotherapy in the treatment of dysthymia. We think therefore that the issue of how many sessions are required needs more research before a statement like this can be made.

Gaudiano et al also refer to the cognitive-behavioral analysis system of psychotherapy (CBASP) for chronic depression. They suggest that the study on CBASP gives evidence that CBT is effective in dysthymia. However, CBASP is a combination of different types of psychotherapy techniques including not only CBT, but also interpersonal, psychodynamic, and behavioral approaches. Furthermore, the study by Keller et al focused on patients with chronic major depression, not dysthymia. No patients with pure dysthymia were included in this study, and therefore it is difficult to see how this study gives evidence that CBASP, let alone CBT, is effective in dysthymia!

So what is the evidence for the statement that “comprehensive CBT adapted for chronic depression is a promising treatment for dysthymia”? We have one study that finds negative findings compared to pharmacotherapy and another study that is not about CBT and not about dysthymia. Would this not meet the definition of “premature conclusions”?

Finally, our last comment is that we do not exclude the possibility that psychotherapy could be a promising treatment for dysthymia. We just do not have the evidence available yet.

References


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