

## Review

# Quantified superiority of cognitive behaviour therapy to antidepressant drugs: a challenge to an earlier meta-analysis

Parker GB, Crawford J, Hadzi-Pavlovic D. Quantified superiority of cognitive behaviour therapy to antidepressant drugs: a challenge to an earlier meta-analysis.

**Objective:** The study aimed to review the conclusion of a previously published meta-analysis which quantified distinct superiority of cognitive therapy to antidepressant drug-therapy ( $P < 0.0001$ ).

**Method:** We sought to include all studies used in the original meta-analysis. Adopting both that study's inclusion criteria and additional criteria resulted in a reduced set of studies. We analysed both 'completer' and 'intention to treat' data, using effect size and odds ratio quantification.

**Results:** There was an overall trend for cognitive therapy to be superior to antidepressant drug-therapy, but this was significant for only one of the four meta-analyses (an intention to treat analysis). We demonstrated considerable heterogeneity between studies, and a significantly higher drop-out rate in the antidepressant groups.

**Conclusion:** The previous interpretation – cognitive therapy being distinctly superior to antidepressant medication – cannot be sustained from the currently analysed data set.

**G. B. Parker<sup>1,2</sup>, J. Crawford<sup>1,2</sup>,  
D. Hadzi-Pavlovic<sup>1,2</sup>**

<sup>1</sup>School of Psychiatry, University of New South Wales and <sup>2</sup>Black Dog Institute, Prince of Wales Hospital, Sydney, Australia

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Gordon B. Parker, Black Dog Institute, Hospital Road, Prince of Wales Hospital, High Street, Randwick NSW 2031, Sydney, Australia.  
E-mail: g.parker@unsw.edu.au

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### Summations

- Our analyses fail to confirm distinctive superiority of cognitive behaviour therapy compared with antidepressant drugs suggested in an earlier meta-analysis.
- Differing conclusions across the previous and current meta-analysis is likely to reflect study inclusion criteria and heterogeneity of results across studies.
- As, in both studies, depressed patients allocated to antidepressant treatment had a significantly higher drop-out rate relative to depressed patients allocated to cognitive therapy, a differential advantage to cognitive therapy would be expected to influence analyses and favour the treatment sustained longer by trial subjects.

### Considerations

- The small number of trials included in our analyses would have reduced the statistical power available.
- As there was significant heterogeneity of results between the trials, and only a small number of studies included in the meta-analyses, one or two studies with very distinctive findings could have impacted on the overall results. The small number of studies included did not permit investigation of sources of the heterogeneity between trials.
- As the aim of this study was to review the conclusion of a previous meta-analysis published in 1998, only trials examined in the previous meta-analysis were included. Hence, the current meta-analyses did not include trials published since 1998.

## Introduction

In 1998, Gloaguen and colleagues published a meta-analysis of the efficacy of cognitive therapy (CT) for depressed patients (1). Those authors focused on four principal comparisons: with CT compared with i) waiting list or placebo, ii) behaviour therapy, iii) other psychotherapies (excluding behaviour therapy) and iv) antidepressant drugs.

The authors concluded that CT was superior to control conditions (wait list or placebo), equal to behaviour therapy, superior to 'other psychotherapies' and superior to antidepressants – with the last difference being highly significant (the authors quoting the probability statistic as  $<0.0001$ ). Such superiority of CT to antidepressant drugs is distinctive, counter-intuitive at first pass, and at variance with our more recent reviews of the published literature in relation to cognitive behaviour therapy (CBT) (2, 3). While an extended period has passed since that meta-analysis was published, there should not be a statute of limitations critiquing on any influential paper.

Our earlier reviews of cognitive behaviour therapy (2, 3) suggested that the efficacy of CBT was highly dependent on the choice of comparator group, and that some of the results from the analysis by Gloaguen and colleagues may have been so confounded. Various findings from the Gloaguen report have been submitted to close consideration in several papers. Wampold and colleagues (4) obtained the data used by Gloaguen and colleagues (1) to re-examine the comparison of CT and 'other psychotherapies'. They then classified each of the 'other psychotherapies' as 'bona fide' or 'non-bona fide' (on the basis of criteria such as 'treatment contained psychologically valid components'), and undertook two separate meta-analyses. The first analysed data from studies making comparisons of CT and 'bona fide' other psychotherapies, whereas the second analysed CT compared with 'non-bona fide' other psychotherapies. Their analyses indicated that while CT was superior to 'non-bona fide' other psychotherapies, it was not superior to 'bona fide' other psychotherapies.

Haby and colleagues (5) focused on comparisons of CBT and control (wait list, placebo or attention/psychological placebo). Although their analyses were not restricted to only the studies included in the Gloaguen et al. analysis, they quantified a similar effect size to that quantified by Gloaguen et al. (i.e. 0.77 and 0.82 respectively), although 'the included studies differ markedly'. Nevertheless, they observed that 'On close examination of the studies included in the Gloaguen meta-analysis, we discovered that many did not fit the stated inclusion criteria

of major depression or dysthymic disorder, according to research diagnostic (RDC) or DSM criteria'.

These two relatively recently published papers answer some questions but sharpen one key issue that was highlighted in the Gloaguen report – its quantification of CT being distinctly superior to antidepressant drugs, both in the short-term and in suggesting differential extended benefits. In relation to the latter, Gloaguen et al. (1) summarized the results of 8 follow-up studies comparing CT and antidepressants at a follow-up period of at least 1 year. They noted that, as there was a small number of studies (i.e. 8) and quite varying lengths of follow-up, they undertook a 'simple comparison of the percentage of relapse' rather than undertake a meta-analysis for this component. They concluded that 'CT demonstrated relapse prevention effects that exceeded those of antidepressants in naturalistic follow-ups ranging from 1–2 years', with respective relapse rates of 29% and 60%. As treatment duration is likely to be a strong confounder of relapse prevention data, we focus only on the first claim put by Gloaguen and colleagues – that CT was demonstrated to be distinctly superior to antidepressant drugs in the short term – and proceeded by re-examining the database used by Gloaguen and colleagues to identify possible determinants of such a distinctive finding. There has been only one similar review of this topic. DeRubies and colleagues (6) conducted a meta-analysis of CBT compared with antidepressants using four studies included in the Gloaguen meta-analysis, and reported a small and non-significant effect size in favour of CBT (effect size of 0.22 for post-treatment mean BDI scores). Their analyses were restricted to subsets of severely depressed patients, and they analysed 'intention to treat' (ITT) rather than 'completer' data.

### Aims of the study

The aim of this study was to review the conclusion of a previous meta-analysis published by Gloaguen and colleagues in 1998 – that cognitive behaviour therapy for depression is distinctly superior to antidepressant drug-therapy at post-treatment. We aimed to re-examine only the trials included by Gloaguen and colleagues.

## Material and methods

### Selection of studies

Of the total 48 studies reported by Gloaguen as being included in their overall meta-analyses, we identified 17 trials (as did Gloaguen et al.) which

included a group receiving cognitive therapy or cognitive behaviour therapy (but not necessarily alone) and a group receiving antidepressant treatment (but not necessarily alone). For example, some studies comparing CT with CT plus an antidepressant. These 17 trials were: i) Beck et al. (7), ii) Beutler et al. (8), iii) Blackburn et al. (9) and Blackburn et al. (10), iv) Bowers (11), v) Covi & Lipman (12), vi) Dunn (13), vii) Elkin et al. (14), viii) Hautzinger & De Jong-Meyer (15), ix) Hollon et al. (16), x) Macaskill & Macaskill (17), xi) McLean & Hakstian (18), xii) Murphy et al. (19) and Simons et al. (20), xiii) Murphy et al. (21), xiv) Rush et al. (22) and Kovacs et al. (23), xv) Rotzer-Zimmer et al. (24), xvi) Scott & Freeman (25) and xvii) Zimmer et al. (26) (where two papers are reported, the second paper refers to a follow-up of the same primary study).

Two of the listed trials (24, 26) were reported in unpublished conference presentations, but we were able to obtain copies of both from Jean Cottraux (co-author of the Gloaguen et al. meta-analysis). For one of the trials (15), we were unable to locate the paper describing the trial, which was reported by Gloaguen et al. as ‘in press’. However, we were able to obtain another publication (by identical authors and published in the same year) reporting trial results.

To test the comparison as stated by Gloaguen and colleagues, we developed a set of inclusion criteria. Firstly, the randomized controlled trial (RCT) should contain a group receiving antidepressant treatment alone (i.e. not in addition to ‘relaxation’, ‘supportive therapy’ or cognitive therapy) and a group receiving cognitive therapy or cognitive behaviour therapy alone (i.e. not in addition to an antidepressant or placebo medication). Secondly, as Gloaguen et al. stated that they used the Beck Depression Inventory (BDI) as ‘the common measure of effectiveness across all the trials’, we only included studies that reported post-treatment results in terms of BDI means and standard deviations or BDI-operationalized percentages of ‘responders’.

Of the 17 trials analysed by Gloaguen et al., we therefore excluded eight from our analysis, with reasons reported in Table 1. Details of the nine trials used in our analyses are reported in Table 2.

**Statistical analyses**

Prior to conducting any meta-analysis, we classified results from each of the included trials as using either a ‘completers only’ or ‘intent-to-treat’ (ITT) methodology. The advantages and disadvantages of using completer data are described by Elkin

Table 1. Trials excluded from current analyses and reason identified

Authors and study	Reasons for exclusion
Beck et al. (7)	No AD alone arm (only AD plus CT)
Beutler et al. (8)	No CT or CBT alone arm (only CT plus placebo or CT plus AD)
Bowers (11)	No CT or CBT alone arm (only CT plus AD)
Covi & Lipman (12)	No AD alone arm (only AD plus CT)
Dunn (13)	No AD alone arm (only AD plus ‘supportive therapy’)
Scott & Freeman (25)	BDI not used
Macaskill & Macaskill (17)	No CT or CBT alone arm (only CT plus AD)
Zimmer et al. (26)	Post-treatment results not reported

AD, antidepressant drug; CT, cognitive therapy; CBT, cognitive behaviour therapy.

et al. (14, p 973): ‘The completer analysis best reflects treatment effects for those patients who have received a full course of treatment, an important focus of any treatment study. The completer analytic strategy does not, however, take into account any possible biases due to differential attrition in the different treatment conditions’. Hence, we chose to conduct separate meta-analyses for results using both completer data and those using ITT data. Some trials included used both types of methodology; hence these trials were included in more than one meta-analysis.

We conducted four meta-analyses – two using completer data and two using ITT data and, for each strategy, we undertook a meta-analysis for studies reporting BDI means and standard deviations, and a second meta-analyses for studies reporting ‘responders’ based on BDI cut-off scores.

More specifically, among those trials reporting completer data, five studies provided BDI means and standard deviations, and we undertook a meta-analysis of those five comparisons. Secondly, raw odds ratios (ORs) were calculated for the seven studies which reported percentages of ‘responders’ amongst the treatment completers, and we undertook a second meta-analysis of those seven comparisons. Amongst those trials reporting ITT data, six studies provided BDI means and standard deviations, and we undertook a meta-analysis of those six comparisons. Finally, we conducted a meta-analysis of six raw ORs calculated for the six studies which reported percentages of ‘responders’ using ITT data.

All meta-analyses were conducted using the software package *META* (version 0.81) by Schwarzer (27), using a random effect size model.

**Results**

Meta-analyses of comparisons amongst treatment completers

Table 3 presents the results of effect sizes (and confidence intervals of effect sizes) of the five

Table 2. Details of nine trials included in the current analyses

Trial (authors and study)	Inclusion criteria	Sample size (completers)	Treatment details	Post-treatment BDI means and SDs	Criteria for 'responder'	Post-treatment percentages of responders
Blackburn et al. (9)	RDC criteria for primary major depressive disorder and minimum BDI score of 14 (mild) (mean BDI score 23–24)	AD: <i>n</i> = 20 CT: <i>n</i> = 22	AD: 'drug of choice', usually amitriptyline or clomipramine CT: mean of 12 weekly sessions of CT	Not reported	50% decrease on BDI or Hamilton after a maximum of 12 weeks	AD: 55.0% CT: 72.7%
Elkin et al. (14)	RDC criteria for primary major depressive disorder and minimum 17-item Hamilton score of 14 (mean BDI 26–27)	AD: <i>n</i> = 36 CBT: <i>n</i> = 37	AD: imipramine plus clinical management CBT: mean of 16 sessions	AD: 6.5 (SD 8.6) CBT: 10.2 (SD 8.7)	BDI ≤ 9 After at least 12 sessions and 15 weeks of treatment	AD: 69% CBT: 65%
Hautzinger & De Jong-Meyer (15)	DSM-III-R criteria for major depression (unipolar, without melancholic) or dysthymic disorder. Minimum BDI score and Hamilton score of 20	AD: <i>n</i> = 37 CBT: <i>n</i> = 51	AD: amitriptyline for 8 weeks CBT: three sessions per week for 8 weeks (i.e. 24 sessions)	Not reported	BDI and Hamilton scores ≤ 9	AD: 32.4% CBT: 41.2%
Hollon et al. (16)	RDC criteria for major depressive disorder, plus minimum BDI score of 20, plus minimum 17-item Hamilton score of 14	AD: <i>n</i> = 32 CBT: <i>n</i> = 16	AD: imipramine CT: mean of 14.9 CT sessions in 12 weeks	AD: 10.5 (SD 9.5) CT: 7.9 (SD 9.5)	BDI ≤ 9	AD: 56% CT: 62%
McLean & Hakstian (18)	Feighner et al.'s criteria for clinical depression, plus two of three minimum scores, including BDI minimum of 23	AD: <i>n</i> = 39 BT: <i>n</i> = 40	AD: amitriptyline BT: 'behaviour therapy', including 'cognitive self-control' and problem solving	AD: 14.14 BT: 9.70 SDs not reported	BDI ≤ 7	AD: 25% BT: 50%
Murphy et al. (19)	Criteria of Feighner et al. for primary, non-bipolar affective disorder, depressed, plus minimum of 20 on BDI plus minimum of 14 on 17-item Hamilton	AD: <i>n</i> = 16 CT: <i>n</i> = 19	AD: nortriptyline CT: CT for 12 weeks	AD: 8.94 (SD 9.12) CT: 9.53 (SD 8.21)	N/A	Not reported
Murphy et al. (21)	Criteria of Feighner et al. for primary, non-bipolar affective disorder, depressed, plus minimum of 14 on BDI plus minimum of 10 on 17-item Hamilton	AD: <i>n</i> = 7 CBT: <i>n</i> = 11	AD: desipramine CBT: maximum of 20 sessions over 12 weeks	AD: 11.86 (SD 6.96) CBT: 6.37 (SD 6.60)	BDI ≤ 9	AD: 29% CBT: 82%
Rotzer-Zimmer et al. (24)	RDC criteria for major depressive disorder, plus minimum BDI score of 20, plus minimum 17-item Hamilton score of 14	AD: <i>n</i> = 11 CBT: <i>n</i> = 18	AD: amitriptyline or maprotyline 'according to clinical indication' CBT: 12 weeks	Means graphed but not reported. SDs not reported	'BDI ≤ 14, 50% decrease'	AD: 25% CBT: 67%
Rush et al. (22) and Kovacs et al. (23)	Criteria of Feighner et al. for depression, plus minimum BDI score of 20, plus minimum 17-item Hamilton score of 14	AD: <i>n</i> = 14 CT: <i>n</i> = 18	AD: imipramine CT: maximum of 12 CT sessions	AD: 13.00 (SD 12.71) CT: 5.94 (SD 5.33)	BDI ≤ 9	AD: 29.4% CT: 83.3%

AD, antidepressant drug; BDI, Beck Depression Inventory; CT, cognitive therapy; CBT, cognitive behaviour therapy; RDC, research diagnostic; SD, standard deviation.

comparisons for which BDI means and standard deviations were reported, using completer only data. Positive effect sizes are in favour of the antidepressant drug (AD), whilst negative effect sizes are in favour of CT/CBT. The meta-analysis of these five comparisons gave a random effects size of  $d = -0.173$  ( $P = 0.463$ ), indicating a small effect size in favour of CT – but not significant. However, there was significant heterogeneity

amongst the five effect sizes in the meta-analysis ( $Q = 9.9$ ;  $P = 0.042$ ).

Table 4 presents the raw ORs of the seven comparisons for which percentages of 'responders' were reported, amongst those who had completed treatment. Here, ORs greater than 1.0 are in favour of CT/CBT and ORs lesser than 1 are in favour of AD. The meta-analysis of these seven comparisons gave a pooled random OR of 1.363 ( $P = 0.477$ ).

Table 3. Effect sizes (and 95% CI for effect sizes) of five comparisons based on BDI means and SDs using completer data

Trial	Effect size $d^*$	95% Confidence interval for ES
Elkin et al. (14)	0.423	-0.041; 0.887
Hollon et al. (16)	-0.269	-0.872; 0.334
Murphy et al. (19)	0.067	-0.599; 0.732
Murphy et al. (21)	-0.725	-1.710; 0.260
Rush et al. (22)	-0.742	-1.467; -0.017
Pooled	-0.173 ( $P = 0.463$ )	-0.638; 0.290

BDI, Beck Depression Inventory.

\*Positive effect sizes favour AD; negative effect sizes favour CT.

Table 4. Odds ratios of seven comparisons based on percentages of 'responders' using completer data

Trial	OR*	95% Confidence interval for OR
Blackburn et al. (9)	2.182	0.602; 7.902
Elkin et al. (14)	0.804	0.232; 2.010
Hautzinger & De Jong-Meyer (15)	0.686	0.283; 1.663
Hollon et al. (16)	1.296	0.379; 4.434
Murphy et al. (21)	11.250	1.193; 106.123
Rotzer-Zimmer et al. (24)	0.167	0.033; 0.854
Rush et al. (22)	9.000	1.724; 46.994
Pooled	1.363 ( $P = 0.477$ )	0.581; 3.201

\*ORs > 1 favour CT; ORs < 1 favour AD.

Hence, this analysis again showed a non-significant difference in favour of CT/CBT. Also consistent with our first meta-analysis using data for completers, there was significant heterogeneity amongst the seven comparisons using ORs ( $Q = 18.2$ ;  $P = 0.006$ ). We repeated this meta-analysis using relative risk (RR) quantification instead of odds ratios, and found similar results, with a pooled RR of 1.114 ( $P = 0.546$ ).

Meta-analyses of comparisons using ITT data

Tables 5 and 6 consider ITT data. Table 5 presents the results of effect sizes (and confidence intervals of effect sizes) of the five comparisons for which BDI means and standard deviations were reported. The meta-analysis of these five comparisons gave a

Table 5. Effect sizes (and 95% CI for effect sizes) of five comparisons based on BDI means and SDs using ITT data

Trial	Effect size $d^*$	95% Confidence interval for ES
Elkin et al. (14)	0.170	-0.195; 0.534
Hollon et al. (16)	-0.107	-0.577; 0.365
Murphy et al. (19)	-0.198	-0.765; 0.370
Murphy et al. (21)	-1.186	-2.130; -0.241
Rush et al. (22)	-0.947	-1.597; -0.296
Pooled	-0.353 ( $P = 0.128$ )	-0.806; 0.101

\*Positive effect sizes favour AD; negative effect sizes favour CT.

Table 6. Odds ratios (ORs) of five comparisons based on percentages of 'responders' using ITT data

Trial	OR*	95% Confidence interval for OR
Elkin et al. (14)	0.870	0.420; 1.803
Hollon et al. (16)	1.162	0.449; 3.005
McLean & Hakstian (18)	3.417	1.413; 8.262
Murphy et al. (21)	22.500	2.603; 194.507
Rush et al. (22)	12.750	2.883; 56.395
Pooled	3.092 ( $P = 0.035$ )	1.081; 8.844

\*ORs > 1 favour CT; ORs < 1 favour AD.

random effects size of  $d = -0.353$  ( $P = 0.128$ ), indicating a non-significant effect size in favour of CT/CBT. However, there was significant heterogeneity amongst the five effect sizes in the meta-analysis ( $Q = 13.3$ ;  $P = 0.010$ ).

Table 6 presents the raw ORs of the five comparisons for which percentages of 'responders' were reported, using ITT data. Again, ORs greater than 1.0 are in favour of CT/CBT and ORs lesser than 1 are in favour of antidepressant medication. The meta-analysis of these five comparison gave a pooled random OR of 3.092 ( $P = 0.035$ ), significantly in favour of CT/CBT. Again there was significant heterogeneity amongst the comparisons using ORs ( $Q = 18.5$ ;  $P = 0.001$ ). When we quantified this meta-analysis using the RR statistic, instead of ORs, similar results were quantified, with a pooled RR of 1.795 ( $P = 0.043$ ).

Rates of attrition

Table 7 presents the rates of attrition for both the CT group and AD groups, for each of the six trials included in our meta-analyses of ITT results. For all six studies, the attrition rate was greater in the AD group, although the difference did not always reach statistical significance. A logistic analysis of the data in Table 7, fitting effects for studies and treatment showed that patients receiving AD had a 2.1 times greater odds of dropping out ( $P < 0.01$ ). However, the degree to which reasons for dropping out were examined, relative to completers, varied between studies. Rush and colleagues (22) reported that all of their drop-outs had a 'not improved' clinical status at the time of termination, according to their BDI scores. Murphy et al. (21) identified BDI scores in the severe range as a predictor of drop-out from the antidepressant group. McLean and Hakstian (18) reported that the reasons for participants dropping out of the antidepressant group included 'unbearable' side-effects and not liking passive treatments, whereas reasons reported for not completing the



Trial	Number (and percentage) of drop-outs				Statistically significant difference*
	CBT/CT		AD		
Elkin et al. (14)	22/59	37.3%	27/62	43.5%	No ( $P = 0.483$ )
Hollon et al. (16)	9/25	36.0%	25/57	43.9%	No ( $P = 0.506$ )
McLean & Hakstian (18)	2/42	4.8%	10/49	20.4%	Yes ( $P = 0.028$ )
Murphy et al. (19)	5/24	20.8%	8/24	33.3%	No ( $P = 0.330$ )
Murphy et al. (21)	0/11	0.0%	5/12	41.7%	Yes ( $P = 0.016$ )
Rush et al. (22)	1/19	5.3%	8/22	36.4%	Yes ( $P = 0.013$ )

\*Based on our Pearson chi-squared analyses using data from the CBT/CT and AD groups only; these may differ from the studies' reported tests of differences between treatment groups in attrition rates, because of varying numbers of treatments groups.

Table 7. Drop-out rates in the CT and AD groups, for each of the 6 trials included in our meta-analyses of ITT data

'behaviour therapy' treatment included wanting to focus on the existential nature of their depression.

## Discussion

Our analyses (using a refined set of studies) challenges Gloaguen et al.'s conclusion that CT is superior to antidepressant drugs in acute phase randomized controlled trials of each treatment modality. When analyses were restricted to those comparator studies quantifying improvement levels in depression scores, completer analyses failed to quantify any significant difference whether effects sizes or ORs were used for quantification. The intention to treat analysis was again non-significant when effect size quantification was undertaken. The only significant analysis was when ORs were used to quantify the intention to treat analyses. Thus, while there was a consistent trend favouring CT above AD, only one of the four meta-analyses quantified a significant difference.

Differences between the conclusions of Gloaguen et al. study and this study are likely to reflect a number of factors. First, in respecting inclusion criteria put by Gloaguen et al., we were unable to find sustainable reasons for including a number of the studies analysed in their data set. Second, all our analyses indicated considerable heterogeneity in CT/AD differences across the included studies, so when meta-analyses are based on a relatively small number of studies, one or two studies with very distinctive findings can impact on the overall result. Third, we quantified a significantly higher drop-out rate in the overall AD group, which would have contributed to the one significant difference found in our study, when ITT analyses are distinctly influenced by differential drop-out rates. We suspect that differential drop-out rates were a key factor contributing to the original meta-analysis results. Finally, it should be acknowledged that the smaller number of comparisons included in our analyses would have reduced the statistical

power available. Whilst Gloaguen and colleagues included 17 comparisons of antidepressants and CT/CBT (with or without additional treatments), our meta-analyses used five to seven comparisons. However, inspection of the magnitude of the pooled effect sizes provides further support for the importance of distinguishing between ITT and 'completers' data. Our pooled effect size of 0.35 when using ITT data, in favour of CT albeit non-significantly, was comparable with Gloaguen et al.'s finding of 0.38. However, when we restricted our comparisons with those amongst treatment completers only, the pooled effect size was a reduced 0.17.

Differences may exist between CT and ADs in their acute efficacy for managing depression. It might be imagined, for example, that antidepressant drugs would be more effective for the 'more severe' depressive disorders or for those with 'more biological' disorders such as melancholia. If true, then studies weighting those with 'less' or 'more' severe disorders, or 'low' or 'high' rates of melancholia could produce quite differing results – respectively quantifying the superiority of CT and, conversely, antidepressant drug. Thus, any inquiry as to whether any psychotherapy is superior, inferior or comparable in its efficacy to an antidepressant drug not only risks logical challenge but can lead to misleading treatment guidelines. Further studies, particularly ones that include comparative CT and AD studies published since the original meta-analysis, would benefit from investigation into the heterogeneity of trial results, examining predictors of response across differing depressive subtypes and levels of depression severity to both CT and antidepressants – as well as examining therapeutic nuances (e.g. drug doses and quality of CT interactions).

We conclude then that the conclusion from the previously reported meta-analysis published by Gloaguen and colleagues in 1998 – indicating striking superiority to cognitive therapy compared with antidepressant drug – is not sustainable.

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## Declaration of Interest

Gordon Parker is Executive Director of Australia's Black Dog Institute and has served on pharmaceutical advisory boards and spoken at meetings convened by drug companies.

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