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Review

Efficacy of psychological interventions for adult PTSD in reducing comorbid depression: systematic review and meta-analysis of randomised controlled trials

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Background

Post-traumatic stress disorder (PTSD) and depression are highly comorbid. A comprehensive meta-analysis on the efficacy of PTSD-specific psychotherapies in reducing comorbid depression is lacking.

Aims

To examine the short-, mid- and long-term efficacy of PTSD-specific psychotherapies in reducing comorbid depression.

Method

We performed a preregistered (Prospero-ID: CRD42023479224) meta-analysis and followed PRISMA guidelines. PsycINFO, MEDLINE, Web of Science and PTSDpubs were searched. Randomised controlled trials (RCTs) examining psychotherapies for PTSD in samples with ≥70% PTSD diagnosis rate, mean age of sample ≥18 years, ≥10 participants per group and reporting of depression outcome data were included in the meta-analysis.

Results

In total, 136 RCTs ($N = 8868$) assessed depression. Most data concerned trauma-focused cognitive behaviour therapy (TF-CBT), followed by eye movement desensitisation and reprocessing and non-trauma-focused and other trauma-focused interventions. At post-treatment, TF-CBT was associated with large reductions in depression relative to passive controls (Hedges' $g = 0.97$, 95% CI 0.80–1.14, $k = 46$ trials) and moderate reductions relative to active controls (Hedges' $g = 0.50$, 95% CI 0.35–0.65, $k = 29$). Effects relative to control conditions were similar across the other interventions. Response rates for comorbid depression were three times higher in psychological interventions relative to passive controls (odds ratio 3.07, 95% CI 1.18–7.94, $k = 4$). In head-to-head comparisons, there was evidence for TF-CBT producing higher short-, mid- and long-term reductions in depression than non-trauma-focused interventions. Results at mid- and long term were generally similar to those at treatment end-point.

Conclusions

PTSD-specific psychotherapies are effective in reducing depression. TF-CBT presented with the highest certainty of results. More long-term data for other interventions are needed. Results are encouraging for clinical practice.

Keywords

PTSD; major depression; comorbidity; psychotherapies; meta-analysis.

Post-traumatic stress disorder (PTSD) is a serious, prevalent and potentially chronic mental disorder.[1–3] The individual and societal burden of PTSD is likely to remain high globally in the face of ongoing mass trauma events such as wars[4] or natural disasters.[5] About half of adults suffering from PTSD also present with comorbid depression.[4,6,7] Five potential explanations for this high comorbidity have been brought forward. First, pre-existing PTSD may increase risk for depression. For instance, avoidance of trauma-related situations may cause substantial and enduring loss of reinforcers (e.g. social support), which in turn may contribute to the development of depression.[8] Second, pre-existing depression may serve as a risk factor for PTSD by potentially increasing the likelihood of trauma exposure and maladaptive coping.[9] Third, comorbidity might be influenced by shared environmental factors, with trauma impacting the development of both conditions.[10] Fourth, comorbidity might be affected by shared genetic vulnerability.[11] Fifth, comorbidity may reflect overlapping nosology, with several overlapping symptoms between PTSD and depression.[12] These potential explanations are not mutually exclusive, and the aetiology of PTSD and comorbid depression is highly idiosyncratic. Crucially, individuals suffering from PTSD and depression (relative to either alone) present with increased suicidality[13] and lower levels of functioning,[14] underscoring the necessity of evaluating treatment options.

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Psychological treatments are recommended as first-line treatment options for both conditions.[15,16] However, clinical uncertainties exist about how to treat individuals with comorbid PTSD and depression. The question arises as to whether addressing one condition through targeted treatment will impact the other condition.[6] Addressing these ambiguities requires careful consideration of scientific evidence. In randomised controlled trials (RCTs) of psychological interventions for adult PTSD, depression is often assessed as a secondary outcome. Summarising data from such RCTs can help clarify uncertainties surrounding treatment priorities. Previous related meta-analyses have focused on specific populations with PTSD. For example, O'Doherty et al focused on rape and sexual assault survivors,[17] whereas Morina et al focused on survivors of mass violence in low- and middle-income countries.[18] A comprehensive meta-analysis covering all RCTs and populations (including all trauma types) is lacking. The present work attempted to fill this gap.

Method

We preregistered the objectives and methodology of the present work (Prospero-ID: CRD42023479224) and followed PRISMA 2020 guidelines.[19]

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Identification and selection of studies

For the timespan from inception to 1 April 2023, we relied on our previous systematic search.[16] For the timespan thereafter, we conducted a new search wave with identical search strategy on 2 January 2024. We conducted multi-field searches utilising search terms for PTSD (e.g. 'post-traumatic stress' OR 'PTSD') and treatment (e.g. 'treatment' OR 'intervention') in MEDLINE, PsycINFO, PTSDpubs and Web of Science (see Supplementary Appendix A in the online Supplementary Material available at https://doi.org/10.1192/bjp.2025.10345). At least two of the four investigators (T.H.H., A.S.L., L.H. and A.K.) independently conducted the systematic literature search. Interrater agreement was high (96%). Discrepancies were discussed among at least three authors until consensus was reached. Furthermore, we screened 63 related review articles (see Supplementary Appendix B in the online Supplementary Material). We also screened all reference lists of included RCTs and the Clinical Trials Database of the US Veterans Affairs National Center for PTSD (https://www.ptsd.va.gov/ptsdrepository/index.asp).

Trials were eligible if they met the following inclusion criteria: (a) RCT; (b) PTSD was the primary treatment target; (c) ≥70% of participants were diagnosed with full PTSD at baseline by means of a clinician-based interview; (d) a psychological intervention was compared with a passive or active control condition, or to another psychological intervention (see categorisation, below); (e) mean age of sample ≥18.0 years; and (f) data of at least 10 participants per arm were reported. No restrictions were made with regard to language, publication format or sample characteristics. We included samples with comorbid disorders providing that inclusion criteria (b) and (c) were met. Studies were excluded if treatments targeted several disorders (e.g. PTSD plus traumatic brain injury or PTSD plus substance use disorders). In line with inclusion criterion (c), RCTs were excluded if subjects entered the trial solely on the basis of a self-report (PTSD) measure. In line with the investigated research question, eligible trials furthermore had to assess depression as an outcome for inclusion iin the meta-analysis. We wanted to include a list of all eligible RCTs and a list of RCTs assessing depression as an outcome, to give an overview of how many RCTs in the field do versus do not assess depression as a secondary outcome.

Coding of trial characteristics

At least two of the four investigators (T.H.H., A.S.L., L.H. and A.K.) independently extracted data from eligible RCTs. Discrepancies were discussed among at least three authors until consensus was reached. We extracted both continuous (i.e. standardised mean differences (SMDs) in depression severity) and dichotomous depression data (i.e. treatment response versus non-response). Data extractions started on 6 February 2024. Missing outcome data were solicited via email to the corresponding authors, with a follow-up reminder sent after 1 month. We received depression data of 20 RCTs via email (see Supplementary Appendix C in the online Supplementary Material). In four RCTs, depression data were reported in insufficient detail but data could not be provided by primary authors following our request, mostly due to unavailability of data (see Supplementary Appendix D in the online Supplementary Material). When data of both interview-/clinician-based and self-report-based measures were reported, the former was prioritised. When outcome data were provided based on both intent-to-treat (ITT) and completer analyses, the former was prioritised.

With regard to dichotomous outcome data, we categorised definitions of treatment response in three subcategories: (a) remission (i.e. losing diagnostic status for depression pre- to post-treatment); (b) response (i.e. at least 50% reduction pre- to post-treatment in depression severity); and (c) any kind of clinically significant improvement in depression pre- to post-treatment (e.g. reliable improvement as calculated with the reliable change index[20]). Because our attempt to perform an isolated review per category was not possible due to lack of data ($k < 4$), we pooled data across categories.

Psychological interventions were grouped into the following five categories, based on the number of published RCTs and theoretical basis of a given intervention:[16] (a) trauma-focused cognitive behaviour therapy (TF-CBT; e.g. prolonged exposure[21]); (b) eye movement desensitisation and reprocessing (EMDR[22]); (c) other trauma-focused psychological interventions not based on CBT or EMDR theoretical basis, such as imagery rescripting;[23] (d) non-trauma-focused psychological interventions (e.g. interpersonal psychotherapy[21]); and (e) multidisciplinary treatments (MDTs; i.e. interventions with elements of multiple categories such as STAIR-modified prolonged exposure[24]). Control conditions were categorised into (a) passive control conditions (e.g. waitlist) and (b) active control conditions (e.g. treatment/care-as-usual). See Supplementary Appendix E in the online Supplementary Material for an overview of all categorisations.

Quality assessment

Risk of bias assessment was based on eight dichotomously scored quality criteria reported by Cuijpers et al,[25] and originally based on Cochrane Collaboration criteria[26] and criteria for assessing the quality of treatment delivery.[27] These criteria have been widely used in meta-analytic psychotherapy research.[28,29] See Supplementary Appendix F in the online Supplementary Material for all criteria and their scoring. Quality sum scores may range from 0 to 8, with higher scores indicating lower risk of bias. Guided by previous research,[16] RCTs were classified as being of high quality if seven or more quality criteria were fulfilled.

Statistical analysis

We conducted all meta-analyses in R version 4.5.0 for Windows (RCore Team City, Vienna, Austria; https://www.r-project.org/) using the metafor package version 4.8-0 for Windows (https://cran.r-project.org/package=metafor).[30] Given that we expected considerable heterogeneity, we conducted random-effects meta-analyses. We set the level of statistical significance for all analyses to *P*-values (two-sided) <0.05. For all analyses, we pooled data only when at least four independent data points were available. We first analysed data across all psychological interventions relative to passive and active control conditions, respectively. For multi-arm trials, the primary comparison (as stated by the authors of the original work) was included to avoid data dependencies. We then analysed data per intervention category. With regard to synthesis of continuous outcome data, we calculated Hedges' *g* values.[31] In line with Cohen's benchmarks,[32] Hedges' *g* may be interpreted as small (0.2), moderate (0.5) or large effects (0.8). We calculated both 95% CI and 95% prediction intervals;[30] when both exclude the null, there is particular certainty in the found effect. With regard to the synthesis of dichotomous outcome data (i.e. treatment response of depression), we followed established gold standard procedures: that is, we pooled prevalences (i.e. response rates) with the inverse of the Freeman–Tukey double-arcsine transformation.[33] To obtain 95% CI for forest plots, we utilised the Agresti–Coull method.[34] We also calculated odds ratios for dichotomous outcome data, with a value above 1 indicating increased odds of treatment response of the experimental condition relative to the comparison group (and vice versa), and an odds ratio of 1 indicating no difference in odds of treatment response. For all analyses, we estimated the heterogeneity of effects via the I^2-statistic and *Q*-statistic. The I^2-statistic provides the percentage of true heterogeneity in effect estimates rather than chance; I^2 may be interpreted as indicating low (25%), moderate (50%) or high (75%) heterogeneity. To control for small study effects, we performed Egger's test[35] when sufficient evidence had

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accumulated ($k \geq 10$). Whenever Egger's test indicated significant asymmetry, we applied the trim-and-fill-method,[36] which adds fictitious data points until symmetry is reached. We performed outlier-adjusted reanalyses whenever at least one outlier was detected. We defined outliers as effects scoring at least 3.3 standard deviations above or below the pooled effect.[37]

Various sensitivity analyses were performed to check whether the results are sensitive to relevant methodological factors or sample characteristics. We performed sensitivity analyses for the following subsets of data: Beck Depression Inventory (BDI) (i.e. any version) data only;[38] data split by treatment format (e.g. individually delivered treatment only); high-quality trials only (see risk of bias assessment); 100% female and 100% male samples only; low- and middle-income country data only; and military samples only. We conducted sensitivity analyses rather than moderator analyses because the variables of interest – such as individually delivered treatment – applied to only a subset of trials. The choice of sensitivity analyses was based on previous (network) meta-analyses in the field of psychotherapies for PTSD.[16,39–41] Meta-regressions for analysis of two continuous potential moderators (i.e. mean age and percentage of participants with comorbid substance abuse/disorder) were performed only when sufficient data ($k \geq 10$) had accumulated.[42]

Results

Selection and characteristics of included studies

See Fig. 1 for the study synthesis. Titles and abstracts of 3347 unique hits were screened following duplicate deletion. Of these, 56 reports were thoroughly checked for eligibility in the full-text screening stage, leading to the inclusion of nine eligible reports on nine RCTs. A further two RCTs were identified via ResearchGate[43,44] and one addition trial[45] by screening reference lists of newly included RCTs. As such, 12 new RCTs were identified and added to the 161 identified in our previous search.[16] Of these 173 eligible RCTs (see Supplementary Appendix G in the online Supplementary Material for their references), 136 RCTs (79%) reported on depression outcomes and were included in the meta-analysis.

These 136 RCTs comprised data from a total of 8868 patients. In total, 124 RCTs (91%) exclusively enrolled patients with full PTSD diagnosis at baseline. The majority of enrolled patients identified as female (62%). Mean age (across the 136 RCTs) was 39 years (s.d. = 9.0), with sample means ranging from 18 to 65 years. Across the 48 RCTs that reported the diagnostic status of depression at pre-treatment, 53% of subjects met depression diagnosis. In most RCTs (82%, $k = 112$), interventions were delivered individually. The most frequently used depression measure was BDI (i.e. any version; 56%, $k = 76$), followed by the Patient Health Questionnaire (i.e. any version; 12%, $k = 16$) and other questionnaires. See Supplementary Appendix H in the online Supplementary Material for a pie chart illustrating the distribution of all depression measures included in the meta-analysis.

Risk of bias assessment

Across all RCTs, mean study quality was 6.08 (s.d. = 1.26), indicating moderate to high overall quality. See Supplementary Appendices I and J in the online Supplementary Material for an overview of per-trial study quality assessments and study characteristics, respectively.

Meta-analyses of short-term efficacy

Meta-analytic results for short-term efficacy at treatment end-point are presented in Table 1; Table 2 provides results for mid- (up to 5 months post-treatment) and long-term efficacy (6–24 months post-treatment). At treatment end-point, psychological interventions were associated with large reductions in comorbid depressive symptoms relative to passive controls ($g = 0.96$, 95% CI 0.80–1.12, $k = 64$). Heterogeneity was high ($I^2 = 78.71$) and highly significant. Egger's test was significant, indicating significant small study effects. Nevertheless, the trim-and-fill method did not add any study. The prediction interval included the null (95% CI −0.16 to 2.09), limiting certainty in results. Results remained very similar when two statistical outliers were excluded ($g = 0.90$, 95% CI 0.76–1.04, $k = 62$). Heterogeneity remained high ($I^2 = 71.69$) and highly significant. Likewise, results remained very similar when limiting analysis to BDI data only, data from trials with fully (i.e. 100%) individual delivery of treatment(s) only, data from high-quality trials only, data from 100% female samples only and data from low- and middle-income countries only, respectively. Notably, in some of these analyses the prediction interval excluded the null, highlighting particular certainty in the observed effect (i.e. sensitivity analysis concerning BDI data only, data from trials with fully individual delivery of treatment only and data from high-quality trials only).

Relative to active control conditions at treatment end-point, psychological interventions overall were associated with moderate reductions in comorbid depressive symptoms ($g = 0.47$, 95% CI 0.36–0.59, $k = 46$). Heterogeneity was moderate ($I^2 = 46.15$) and highly significant. The prediction interval included the null (95% CI −0.05 to 0.99), limiting certainty in results. Results remained similar when the trim-and-fill method added 13 trials to the left to correct for significant small study effects ($g = 0.31$, 95% CI 0.18–0.44, $k = 59$). Note that the trim-and-fill method does not supply prediction intervals. When limiting analysis to BDI data only, data from trials with individual delivery of treatment(s) only, data from high-quality trials only, data from 100% female samples only, data from 100% male samples only and data from low- and middle-income countries only, results remained very similar, respectively. That is, psychotherapies mostly produced moderate short-term reductions in comorbid depression relative to active control conditions. While certainty in results was limited in some analyses, as illustrated by the prediction interval including the null, some prediction intervals excluded the null (i.e., BDI data only, data from 100% male samples only and data from low- and middle-income countries only). Notably, in studies conducted in low- and middle-income countries, psychotherapies produced large (not moderate) reductions in comorbid depressive symptoms relative to active control conditions ($g = 0.85$, 95% CI 0.45–1.24, $k = 7$). Heterogeneity was moderate ($I^2 = 53.47$) and significant in this sensitivity analysis. No outlier was observed. Of the 136 RCTs reporting depression data, 24 (18%) involved military samples. Sensitivity analyses were feasible for treatment end-point data only (i.e. $k < 4$ for mid- and long-term data). In military samples, psychological interventions compared with active control conditions also yielded significant short-term reductions in comorbid depression ($g = 0.41$, 95% CI 0.24–0.58, $k = 15$). Results remained very similar when one statistical outlier was excluded ($g = 0.37$, 95% CI 0.21–0.53, $k = 14$).

Most accumulated data pertained to TF-CBT, followed by (in the following order) non-trauma-focused interventions, EMDR, other trauma-focused interventions and MDTs. Too few trials investigated MDTs to warrant any MDT-specific review. Results for TF-CBT only were similar to the overarching analyses: that is, large effects were found relative to passive controls ($g = 0.97$, 95% CI 0.80–1.14, $k = 46$) and moderate effects relative to active controls ($g = 0.50$, 95% CI 0.35–0.65, $k = 29$), and these results remained similar across sensitivity analyses (see Table 1). Certainty in effects of TF-CBT was high in various analyses, as highlighted by several prediction intervals excluding the null.

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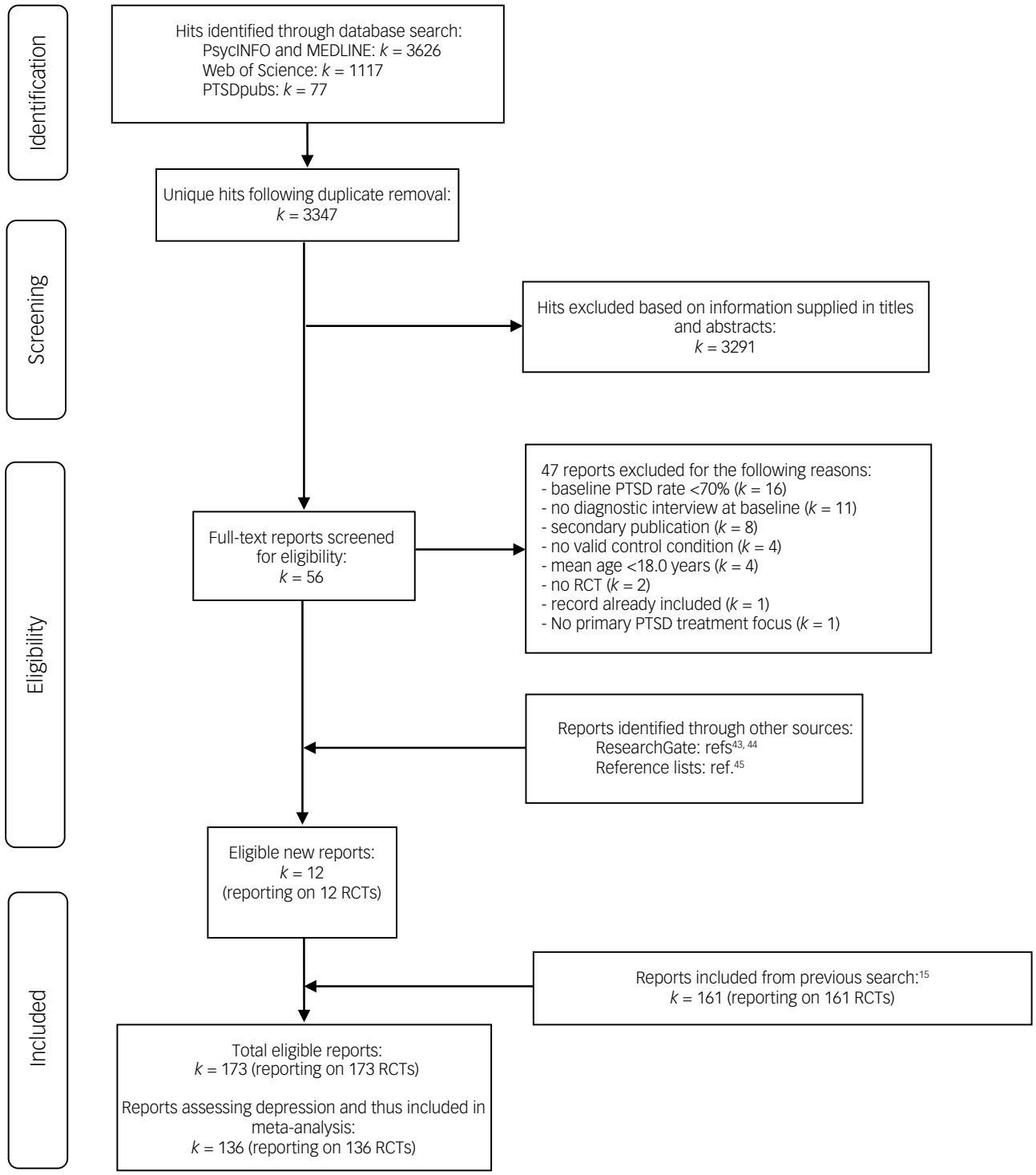

Fig. 1 PRISMA flowchart of study selection. PTSD, post-traumatic stress disorder; RCT, randomised control trial.

Results of EMDR, other trauma-focused interventions and non-trauma-focused interventions were based on considerably less available evidence than that for TF-CBT. EMDR showed large and moderate short-term effects in reducing comorbid depression when compared with passive control conditions ($g = 0.89$, 95% CI 0.43–1.35, $k = 8$) and active control conditions ($g = 0.50$, 95% CI 0.26–0.75, $k = 9$), respectively. With regard to lack of trials, only two sensitivity analyses were feasible (i.e. BDI data only and data from trials with individual delivery of treatment only). Results remained similar to the overarching results. Various prediction intervals excluded the null, highlighting certainty in the short-term effects of EMDR.

Data for other trauma-focused interventions were limited ($k = 4$), allowing only one comparison with passive control conditions, which demonstrated moderate short-term efficacy ($g = 0.66$, 95% CI 0.37–0.94, $k = 4$). The prediction interval excluded the null, highlighting certainty in effect. Sensitivity analyses were infeasible for other trauma-focused interventions due to lack of trials.

Large short-term effects in reducing depression were found for non-trauma-focused interventions compared with passive control conditions ($g = 1.02$, 95% CI 0.52–1.52, $k = 15$), and small significant short-term effects compared with active control conditions ($g = 0.30$,

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Table 1 Short-term efficacy of psychological interventions for adult PTSD in reducing comorbid depression

Comparison of therapies	Number of trials, *k*	Hedges' *g*	95% CI [95% prediction interval]	*P*	I^2
All interventions relative to passive control conditions					
All interventions versus passive control conditions	64	0.96***	0.80 to 1.12 [−0.16 to 2.09]	<0.001	78.71***
All interventions versus passive control conditions (outlier-adjusted analysis)	62	0.90***	0.76 to 1.04 [−0.02 to 1.82]	<0.001	71.69***
All interventions versus passive control conditions – BDI only	38	0.96***	0.77 to 1.15 [−0.04 to 1.95]	<0.001	74.61***
All interventions versus passive control conditions – BDI only (outlier-adjusted analysis)	37	**0.92*** **	**0.75 to 1.10 [0.03 to 1.82]**	<0.001	70.78***
All interventions versus passive control conditions – individual treatment only	53	1.03***	0.85 to 1.20 [−0.10 to 2.15]	<0.001	79.28***
All interventions versus passive control conditions – individual treatment only (outlier-adjusted analysis)	51	**0.95*** **	**0.80 to 1.10 [0.07 to 1.83]**	<0.001	70.45***
All interventions versus passive control conditions – high-quality trials only	30	0.98***	0.77 to 1.18 [−0.02 to 1.97]	<0.001	78.30***
All interventions versus passive control conditions – high-quality trials only (outlier-adjusted analysis)	29	**0.91*** **	**0.75 to 1.07 [0.23 to 1.58]**	<0.001	62.70***
All interventions versus passive control conditions – female only	17	1.19***	0.75 to 1.63 [−0.57 to 2.95]	<0.001	89.55***
All interventions versus passive control conditions – LMICs only	13	1.20***	0.68 to 1.71 [−0.61 to 3.00]	<0.001	90.00***
All interventions relative to active control conditions					
All interventions versus active control conditions	46	0.47***	0.36 to 0.59 [−0.05 to 0.99]	<0.001	46.15***
All interventions versus active control conditions (trim-and-fill adjusted[a])	59	0.31***	0.18 to 0.44	<0.001	64.86***
All interventions versus active control conditions – BDI only	28	**0.65*** **	**0.51 to 0.79 [0.24 to 1.07]**	<0.001	30.13
All interventions versus active control conditions – individual treatment only	39	0.49***	0.36 to 0.62 [−0.07 to 1.06]	<0.001	48.69***
All interventions versus active control conditions – individual treatment only (trim-and-fill adjusted[a])	50	0.33***	0.18 to 0.47	<0.001	65.62***
All interventions versus active control conditions – high-quality trials only	18	0.39***	0.20 to 0.58 [−0.26 to 1.04]	<0.001	63.77***
All interventions versus active control conditions – female only	9	0.57***	0.30 to 0.84 [−0.03 to 1.18]	<0.001	46.78
All interventions versus active control conditions – male only	4	**0.64*** **	**0.29 to 0.99 [0.29 to 0.99]**	<0.001	0.00
All interventions versus active control conditions – LMICs only	7	**0.85*** **	**0.45 to 1.24 [0.01 to 1.69]**	<0.001	53.47*
All interventions versus active control conditions – military samples only	15	0.41***	0.24 to 0.58 [−0.00 to 0.82]	<0.001	33.90
All interventions versus active control conditions – military samples only (outlier-adjusted analysis)	14	**0.37*** **	**0.21 to 0.53 [0.04 to 0.70]**	<0.001	23.52
TF-CBT relative to passive control conditions					
TF-CBT versus passive control conditions	46	0.97***	0.80 to 1.14 [−0.01 to 1.95]	<0.001	74.79***
TF-CBT versus passive control conditions – BDI only	29	**0.99*** **	**0.78 to 1.21 [0.02 to 1.97]**	<0.001	74.28***
TF-CBT versus passive control conditions – individual treatment only	41	**0.98*** **	**0.80 to 1.16 [0.00 to 1.96]**	<0.001	74.87***
TF-CBT versus passive control conditions – high-quality trials only	22	**0.91*** **	**0.73 to 1.09 [0.24 to 1.59]**	<0.001	63.57***
TF-CBT versus passive control conditions – high-quality trials only (outlier-adjusted analysis)	21	**0.85*** **	**0.71 to 1.00 [0.41 to 1.30]**	<0.001	42.87*
TF-CBT versus passive control conditions – female only	11	1.30***	0.77 to 1.84 [−0.43 to 3.03]	<0.001	89.11***
TF-CBT versus passive control conditions – LMICs only	8	1.18***	0.66 to 1.71 [−0.22 to 2.58]	<0.001	82.49***
TF-CBT relative to active control conditions					
TF-CBT versus active control conditions	29	0.50***	0.35 to 0.65 [−0.08 to 1.08]	<0.001	49.92***
TF-CBT versus active control conditions – BDI only	18	**0.67*** **	**0.49 to 0.85 [0.24 to 1.10]**	<0.001	28.24
TF-CBT versus active control conditions – individual treatment only	27	0.49***	0.33 to 0.65 [−0.09 to 1.07]	<0.001	49.96**
TF-CBT versus active control conditions – high-quality trials only	12	0.47***	0.21 to 0.76 [−0.32 to 1.27]	<0.001	71.67***

(Continued)

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Table 1 (*Continued*)

Comparison of therapies	Number of trials, *k*	Hedges' *g*	95% CI [95% prediction interval]	*P*	I^2
TF-CBT versus active control conditions – female only	6	**0.70*** **	**0.39 to 1.01 [0.16 to 1.24]**	<0.001	34.42
TF-CBT versus active control conditions – LMICs only	5	**0.89*** **	**0.43 to 1.35 [0.03 to 1.76]**	<0.001	50.57
TF-CBT versus active control conditions – military samples only	8	0.40***	0.17 to 0.63 [−0.05 to 0.86]	<0.001	37.45
EMDR relative to passive control conditions					
EMDR versus passive control conditions	8	0.89***	0.43 to 1.35 [−0.34 to 2.12]	<0.001	78.70***
EMDR versus passive control conditions – BDI only	4	0.71	−0.03 to 1.46 [−0.85 to 2.28]	0.060	86.83***
EMDR versus passive control conditions – individual treatment only	7	**1.03*** **	**0.63 to 1.44 [0.08 to 1.99]**	<0.001	68.00**
EMDR relative to active control conditions					
EMDR versus active control conditions (all individual treatments in face-to-face format)	9	**0.50*** **	**0.26 to 0.75 [0.04 to 0.97]**	<0.001	28.17
EMDR versus active control conditions – BDI only	7	**0.67*** **	**0.43 to 0.91 [0.43 to 0.91]**	<0.001	0
Other-TF-PIs relative to passive control conditions					
Other-TF-PIs versus passive control conditions (all individual treatment and high-quality trials)	4	**0.66*** **	**0.37 to 0.94 [0.37 to 0.94]**	<0.001	0
Non-TF-PIs relative to passive control conditions					
Non-TF-PIs versus passive control conditions	15	1.02***	0.52 to 1.52 [−0.85 to 2.89]	<0.001	89.89***
Non-TF-PIs versus passive control conditions – BDI only	9	1.01***	0.45 to 1.58 [−0.61 to 2.64]	<0.001	85.81***
Non-TF-PIs versus passive control conditions – individual treatment only	10	1.25***	0.51 to 2.00 [−1.11 to 3.61]	<0.001	93.80***
Non-TF-PIs versus passive control conditions – high-quality trials only	5	1.11*	0.05 to 2.18 [−1.43 to 3.66]	0.040	95.51***
Non-TF-PIs versus passive control conditions – female only	6	1.09*	0.17 to 2.01 [−1.26 to 3.44]	0.020	92.51***
Non-TF-PIs relative to active control conditions					
Non-TF-PIs versus active control conditions	12	**0.30*** **	**0.15 to 0.45 [0.05 to 0.55]**	<0.001	14.33
Non-TF-PIs versus active control conditions (outlier-adjusted analysis)	11	**0.27*** **	**0.13 to 0.42 [0.09 to 0.46]**	<0.001	6.64
Non-TF-PIs versus active control conditions – BDI only	5	0.47**	0.15 to 0.79 [−0.09 to 1.03]	0.004	41.98
Non-TF-PIs versus active control conditions – individual treatment only	7	0.31*	0.03 to 0.60 [−0.28 to 0.90]	0.031	50.84*
Non-TF-PIs versus active control conditions – high-quality trials only	4	0.26	−0.04 to 0.55 [−0.23 to 0.75]	0.092	42.98
Non-TF-PIs versus active control conditions – military samples only	6	**0.35*** **	**0.14 to 0.55 [0.07 to 0.62]**	<0.001	13.12
Head-to-head comparisons (directly compared within RCTs)					
TF-CBT versus EMDR (all individual treatments in face-to-face format)	9	−0.08	−0.44 to 0.27 [−0.94 to 0.77]	0.642	57.37*
TF-CBT versus EMDR – BDI only	7	−0.05	−0.47 to 0.38 [−1.01 to 0.91]	0.825	62.16*
TF-CBT versus non-TF-PIs	22	0.10	−0.01 to 0.22 [−0.26 to 0.47]	0.083	42.37*
TF-CBT versus non-TF-PIs – BDI only	11	**0.20****	**0.08 to 0.33 [0.08 to 0.33]**	<0.001	0
TF-CBT versus non-TF-PIs – BDI only (outlier-adjusted analysis)	10	**0.22*** **	**0.10 to 0.35 [0.10 to 0.35]**	<0.001	0
TF-CBT versus non-TF-PIs – individual treatment only	18	0.13*	0 to 0.25 [−0.19 to 0.44]	0.047	31.43
TF-CBT versus non-TF-PIs – individual treatment only (outlier-adjusted analysis)	17	0.14*	0.02 to 0.26 [−0.14 to 0.43]	0.021	28.11
TF-CBT versus non-TF-PIs – high-quality trials only	14	0.08	−0.07 to 0.22 [−0.32 to 0.47]	0.316	49.27*
TF-CBT versus non-TF-PIs – military samples only	7	0.12	−0.05 to 0.29 [−0.18 to 0.42]	0.179	27.89

PTSD, post-traumatic stress disorder; BDI, Beck Depression Inventory; LMICs, low- and middle-income countries; TF-CBT, ttrauma-focused cognitive behaviour therapy; EMDR, eye movement desensitisation and reprocessing; TF-PIs, trauma-focused psychological interventions; BDI-only trials assessed depression severity with any version of BDI (i.e. BDI, BDI-II or BDI-13); high-quality trials only, analysis including trials fulfilling at least seven of eight quality criteria; I^2, estimate of between-study heterogeneity: asterisks denote the level of statistical significance of the corresponding *Q*-statistic; individual treatment only, trials with individual delivery of intervention (i.e. group or mixed formats excluded); non-TF-PIs, any psychological interventions not applying a trauma focus in treatment; other-TF-PIs, any trauma-focused psychological interventions not based on CBT or EMDR theoretical framework; *P*, *P*-value of the random-effects meta-analysis; trim-and-fill adjusted, trim-and-fill method was used to adjust for small study effects.
Bold text indicates that both 95% CI and 95% prediction interval excluded the null, indicating particular statistical certainty in the significance of the respective result. A positive standardised mean difference favours the psychological intervention (relative to the reference group), and vice versa.
a. The trim-and-fill method does not supply prediction intervals.
*$P < 0.05$, **$P < 0.01$, ***$P < 0.001$.

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Table 2 Mid- and long-term efficacy of psychological interventions for adult PTSD in reducing comorbid depression

Comparison of therapies	Number of trials, *k*	Hedges' *g*	95% CI [95% prediction interval]	*P*	I^2
Mid-term efficacy (i.e. assessments ≤5 months post-treatment)					
All interventions versus passive control conditions	15	0.65***	0.28 to 1.01 [−0.68 to 1.97]	0.001	80.71***
All interventions versus active control conditions	24	0.30***	0.15 to 0.45 [−0.19 to 0.78]	<0.001	42.26*
TF-CBT versus passive control conditions	9	0.72*	0.13 to 1.30 [−1.02 to 2.46]	0.017	87.52***
TF-CBT versus active control conditions	18	0.37***	0.19 to 0.55 [−0.13 to 0.87]	<0.001	41.05*
Non-TF-PIs versus active control conditions	5	0.06	−0.25 to 0.37 [−0.50 to 0.62]	0.707	47.07
TF-CBT versus EMDR	4	−0.33	−0.86 to 0.19 [−1.18 to 0.51]	0.210	40.61
TF-CBT versus non-TF-PIs	13	0.15	−0.01 to 0.31 [−0.25 to 0.56]	0.063	45.28*
TF-CBT versus non-TF-PIs (outlier-adjusted analysis)	12	0.18*	0.02 to 0.34 [−0.20 to 0.56]	0.026	43.18
Long-term efficacy (i.e. assessments 6–24 months post-treatment)					
TF-CBT versus active control conditions	15	0.51***	0.30 to 0.71 −0.06 to 1.07	<0.001	45.07*
TF-CBT versus non-TF-PIs	14	0.18*	0.04 to 0.32 −0.16 to 0.52	0.014	36.89
TF-CBT versus non-TF-PIs (trim-and-fill adjusted[a])	18	0.09	−0.05 to 0.24	0.201	45.45*

PTSD, post-traumatic stress disorder; EMDR, eye movement desensitisation and reprocessing; I^2, estimate of between-study heterogeneity: asterisks denote the level of statistical significance of the corresponding *Q*-statistic; TF-CBT, trauma-focused cognitive behaviour therapy; non-TF-PIs, non-trauma-focused psychological interventions (i.e. any psychological intervention not applying a trauma focus in treatment); *P*, *P*-value of the random-effects meta-analysis; individual treatments, trials with individual delivery of intervention; trim-and-fill adjusted, trim-and-fill-method was used to adjust for small study effects. A positive standardised mean difference favours the psychological intervention (relative to the reference group), and vice versa.
a. The trim-and-fill method does not supply prediction intervals.
*$P < 0.05$, ***$P < 0.001$.

95% CI 0.15–0.45, $k = 12$). Prediction intervals included the null for comparison with passive control conditions, whereas the prediction interval for comparison with active control conditions excluded the null. Some sensitivity analyses were possible. While results were mostly significant and similar to the overarching analysis, prediction intervals mostly excluded the null, limiting certainty in effects. Notably, the sensitivity analysis on high-quality trials revealed no significant short-term reductions in depression relative to active control conditions ($g = 0.26$, 95% CI −0.04 to 0.55, $k = 4$).

In head-to-head comparison, the efficacy of TF-CBT and EMDR did not differ significantly, including a sensitivity analysis on BDI data only. TF-CBT was, however, associated with larger short-term reductions in depression than non-trauma-focused interventions in two sensitivity analyses (i.e. BDI data only and data from trials with individual delivery of treatment only).

Meta-analyses of mid-term efficacy

At mid-term assessment (up to 5 months post-treatment), psychological interventions overall were associated with moderate reductions in depression relative to passive controls ($g = 0.65$, 95% CI 0.28–1.01, $k = 15$). Heterogeneity was large ($I^2 = 80.71$). No outliers or small study effects were detected. Psychological interventions were associated with small mid-term reductions in comorbid depression relative to active controls ($g = 0.30$, 95% CI 0.15–0.45, $k = 24$). Heterogeneity was moderate ($I^2 = 42.26$). No outliers or small study effects were detected. Only TF-CBT and non-trauma-focused intervention showed sufficient mid-term evidence for isolated analysis relative to controls. While the results of TF-CBT were very similar to the overarching analysis, non-trauma-focused interventions did not yield significant reductions in comorbid depression relative to active control conditions ($g = 0.06$, 95% CI −0.25 to 0.37, $k = 5$). Heterogeneity for the mid-term effects of non-trauma-focused interventions was moderate ($I^2 = 47.07$) but non-significant. No outliers were detected. TF-CBT and EMDR did not differ significantly in efficacy at mid-term; however, TF-CBT yielded higher mid-term reductions for depression than non-trauma-focused interventions in the outlier-adjusted analysis ($g = 0.18$, 95% CI 0.02–0.34, $k = 12$). No significant difference was observed in the main analysis ($g = 0.15$, 95% CI −0.01 to 0.31, $k = 13$).

Meta-analyses of long-term efficacy

At long-term assessment (6–24 months post-treatment), only TF-CBT had sufficient available data relative to control conditions. TF-CBT produced moderate reductions in depressive symptoms compared with active controls ($g = 0.51$, 95% CI 0.30–0.71, $k = 15$). Heterogeneity was moderate ($I^2 = 45.07$) and no outliers or small study effect were detected. The only head-to-head comparison with sufficient data concerned TF-CBT relative to non-trauma-focused interventions, with the former producing superior long-term reduction in depression in the main analysis ($g = 0.18$, 95% CI 0.04–0.32, $k = 14$) but not in the trim-and-fill-adjusted analysis ($g = 0.09$, 95% CI −0.05 to 0.24, $k = 18$).

Moderator results

Moderator results are provided in Table 3. In trials comparing psychological interventions with passive control conditions, mean age was significantly negatively associated with (short-term) reductions in comorbid depressive symptoms ($k = 63$, $b = -0.02$, $P = 0.044$). The remaining heterogeneity was large (estimate of between-study heterogeneity remaining (rem. I^2) = 77.75). Nevertheless, this moderation was not found significant in trials comparing with active controls, nor in any of the sub-analyses per intervention category. Only 26 out of the 136 RCTs (19%) reported the percentage of participants presenting with comorbid substance abuse/disorder. Only one moderator analysis was feasible ($k = 10$) for the comparison of psychological interventions and passive control conditions at treatment end-point. The percentage of participants with comorbid substance use abuse/disorder was not found to be a significant

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Table 3 Meta-regression analyses for short-term efficacy data

Comparison of therapies	Number of trials, *k*	b_1	*P*	rem. I^2
Analysed moderator: mean age				
All interventions versus passive control conditions	63	**−0.02***	**0.044**	77.75***
All interventions versus active control conditions	44	−0.01	0.236	40.72**
TF-CBT versus passive control conditions	45	−0.01	0.315	74.87***
TF-CBT versus active control conditions	27	−0.01	0.315	44.65**
Non-TF-PIs versus passive control conditions	15	−0.04	0.118	88.71***
Non-TF-PIs versus active control conditions	12	<0.01	0.838	23.07
TF-CBT versus non-TF-PIs	22	<−0.01	0.524	42.10*
Analysed moderator: percentage of participants with comorbid substance abuse/disorder				
All interventions versus passive control conditions	10	0.01	0.613	0

b_1, slope; non-TF-PIs, non-trauma-focused psychological interventions (i.e. any psychological intervention not applying a trauma focus in treatment); *P*, *P*-value of the respective moderator analysis (i.e. meta-regression); rem. I^2, estimate of between-study heterogeneity remaining (i.e. when analysed moderator was accounted for); TF-CBT, trauma-focused cognitive behaviour therapy.
Bold text indicates statistical significance of the respective moderator.
*$P < 0.05$, **$P < 0.01$, ***$P < 0.001$.

Table 4 Rates of treatment response pre- to post-treatment concerning comorbid depression

Comparison of therapies	Number of trials, *k*	Response rate (%)	τ^2	95% CI	I^2	Number of trials, *k*	Odds ratio	95% CI	*P*	I^2
All interventions versus passive	4	72.81	<0.01	59.70 to 84.40	0.0	4	**3.07***	**1.18 to 7.94**	0.021	18.62
control conditions	4	22.34	0.02	5.23 to 44.89	49.3					
TF-CBT versus non-TF-PIs	6	44.90	<0.01	38.01 to 51.88	45.9	6	1.05	0.70 to 1.56	0.825	26.41
	6	43.02	0.03	28.49 to 58.15	83.2***					
TF-CBT versus non-TF-PIs (outlier-	5	42.96	0	36.35 to 49.69	0	5	0.96	0.66 to 1.40	0.823	17.23
adjusted analysis)	5	45.25	0.03	28.85 to 62.16	85.8***					

k, number of independent trials included in the analysis for the given comparison; I^2, estimate of between-study heterogeneity: asterisks denote the level of statistical significance of the corresponding *Q*-statistic; non-TF-PIs, non-trauma-focused psychological interventions (i.e. any psychological intervention not applying a trauma focus in treatment); *P*, *P*-value of odds ratio; TF-CBT, trauma-focused cognitive behaviour therapy.
Bold text indicates statistical significance of the respective odds ratio (i.e. significant difference in odds of treatment response between the first-mentioned psychological interventions and the given comparator).
*$P < 0.05$, ***$P < 0.001$.

moderator of treatment efficacy in alleviating depressive symptoms in the short term ($k = 10$, $b = 0.01$, $P = 0.613$, rem. $I^2 = 0$).

Treatment response rates

Results concerning rates of treatment responders are provided in Table 4. Treatment response data in terms of comorbid depression were very scarce. In trials comparing psychological interventions with passive control conditions, 73% (95% CI 60–84%, $k = 4$) of individuals responded to treatment in terms of depression, whereas only 22% (95% CI 5–45%, $k = 4$) presented with spontaneous depression response in the waitlist conditions. No outliers were detected. Definitions of treatment response varied across these four trials. While three trials[46–48] reported remission rates, one[49] used the reliable change index[20] and reported the rate of reliable improvers. The number of included trials was too small to check for small study effects ($k < 10$). The odds of treatment response in terms of depression were three times larger in psychotherapy conditions relative to passive control conditions (odds ratio 3.07, 95% CI 1.18–7.94, $k = 4$). Heterogeneity was low ($I^2 = 18.62$). Response rates of depression between TF-CBT and non-trauma-focused interventions did not differ significantly in the main analysis, nor when one outlier was removed.

Discussion

To our knowledge, this is the first comprehensive meta-analysis of the efficacy of psychological interventions for adult PTSD in reducing comorbid depression. At baseline, about half of the participants (53%) presented with clinical levels of comorbid depression. Psychological interventions compared with passive controls produced large effects in terms of alleviating comorbid depression, and moderate effects compared with active controls. These results were found at short term (treatment end-point), mid-term (up to 5 months follow-up) and long term (6–24 months follow-up), as well as across various sensitivity analyses. Most available data concerned TF-CBT, which yielded effects with high statistical certainty (i.e. multiple prediction intervals excluded the null, and sensitivity analyses on high-quality trials yielded very similar results to the overarching analyses) and with very similar results across various contexts (i.e. individual delivery of treatment only, low- and middle-income country data only, 100% female samples only and military samples only). There was also evidence for significant short-term efficacy of EMDR, other trauma-focused interventions and non-trauma-focused interventions. Nevertheless, mid- and long-term data were too scarce for an isolated review. The efficacy of TF-CBT and EMDR did not differ at short and mid-term (and insufficient data at long term). TF-CBT appeared superior to non-trauma-focused interventions across assessment periods (i.e. short, mid- and long term) in some analyses. In a limited number of trials reporting on depression treatment response, psychological interventions were associated with about threefold higher odds of treatment response in terms of depression compared with passive controls. We found one significant moderation: in trials comparing with passive controls, age was significantly negatively associated with efficacy ($k = 63$, $b = -0.02$, $P = 0.044$), suggesting that younger participants benefit more from treatment. However, this moderation was found in only one of seven analyses and future research needs to test whether efficacy is indeed moderated by age. Furthermore, we did not find evidence for a moderating effect of

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the percentage of participants presenting with comorbid substance abuse/disorder.

Comparisons with previous literature

The present results confirm and extend previous results. They are similar to those of O'Doherty et al,[17] who reported a large effect of psychological interventions in reducing comorbid depressive symptoms relative to passive control conditions in survivors of sexual trauma (SMD 0.82, 95% CI 0.48–1.17, $k = 12$). While O'Doherty et al found no significant difference in efficacy between TF-CBT and non-trauma-focused interventions (SMD 0.21, 95% CI −0.12 to 0.54, $k = 9$), we found superior effects of TF-CBT across assessment periods. Due to the much larger number of included RCTs, the present work has increased statistical power to detect potential effects. Effect sizes found in the present work were also similar to those of Morina et al,[18] who reported large effects of psychological interventions in reducing depression relative to control conditions in survivors of mass violence in low- and middle-income countries (SMD 0.86, 95% CI 0.64–1.18, $k = 11$). The magnitude of effects found in the present work was also similar to the meta-analysis by Ronconi et al.[50] Notably, effect sizes in the present work were somewhat higher than those found in meta-analyses of depression-specific interventions.[51] This discrepancy may be attributed to participants in PTSD-focused trials having fewer depressive symptoms initially and primarily suffering from PTSD. However, such interpretations remain speculative and various other factors might (also) be different between PTSD- and depression-specific psychotherapy research.

Implications for clinical practice

The present findings are based on the first comprehensive meta-analysis examining the efficacy of PTSD treatments for comorbid depression and are encouraging for clinical practice, because they highlight that psychological interventions for adult PTSD can effectively reduce comorbid depression. Notably, the results remained consistent when analyses were restricted to high-quality trials, which is in line with previous meta-analytic research on PTSD.[28] This consistency strengthens the certainty of the current evidence base. Taken together, this review suggests that TF-CBT in particular should be prioritised for the treatment of PTSD and comorbid depression, given its significant efficacy and high statistical certainty based on a substantial evidence base. It should be noted that most included trials excluded subjects with acute suicidality, which is often associated with severe depression. As such, the current results do not generalise to acutely suicidal individuals with PTSD and depression. Additionally, the included trials in this meta-analysis were aimed at treating subjects with PTSD as the primary diagnosis. Accordingly, our findings suggest that, only in patients with a primary diagnosis of PTSD (and comorbid depression/depressive symptoms), treatment of PTSD is likely to significantly reduce depression. Importantly, our results do not allow for solid personalised (i.e. patient-specific) treatment planning or prognosis. During the development of comprehensive treatment plans for those with comorbid PTSD and depression, it is essential to prioritise addressing the condition causing the greatest distress and functional impairment. As such, treatment planning and prognosis are to be tailored to the individual. While our findings indicate that PTSD-specific treatments on average (i.e. at the group level) led to significantly reduced severity of depression in samples with primary PTSD, it remains crucial to monitor patient outcomes individually in clinical practice and to treat them with depression-specific interventions in case depression persists following PTSD-specific treatment).

Implications for future research

In head-to-head comparisons, TF-CBT demonstrated superiority when compared with non-trauma-focused interventions. This contradicts the Dodo bird verdict,[52] which postulates that all psychological interventions are similar in terms of their efficacy. Notably, EMDR, non-trauma-focused interventions and other trauma-focused interventions also showed significant short-term reductions in comorbid depression relative to control conditions. However, the lack of long-term data and limited reporting on treatment response rates highlight the need for ongoing research, particularly for interventions other than TF-CBT. To date, long-term data remain limited from which to draw firm conclusions about their long-term efficacy. More research is also needed to determine the extent to which psychological interventions targeting depression produce significant benefits for comorbid PTSD symptomatology (i.e. our research question in reverse). Future trials on PTSD should provide more comprehensive reporting on comorbid depression across all assessment time points, including baseline. Only 48 of 136 RCTs (35%) reported the diagnostic status of depression at pre-treatment. While this enabled determination of the pooled baseline comorbidity rate (i.e. of 53% of patients across the 48 RCTs who suffered comorbid PTSD and depression), the trial authors did not specify the severity of depression. As such, it remains unknown how many patients experienced mild, moderate or severe depression. This information would have been valuable in interpretating the findings, particularly regarding their implications for clinical practice.

Strengths of the present work

The present study has several strengths. To our knowledge, it presents by far the largest quantitative review concerning the research question at hand. While previous reviews had a rather narrow focus, our work applied a broad focus across all psychological interventions without any restrictions regarding sample or study characteristics. By means of our broad search covering literature published up to January 2024, we were able to identify a very large number of eligible RCTs ($k = 173$). For 20 RCTs with insufficient reporting of depression outcome data, we were able to receive missing data via email, whereas for four RCTs no data could be sent to us upon request. Accordingly, we were able to include most of the RCTs ($k = 136$) in the meta-analysis (while the remaining RCTs did not assess or report depression data), maximising the power and generalisability of results. In addition to overarching analyses across all data (e.g. all types of interventions, all depression outcome measures, any samples) that presented with highest statistical power but also with highest heterogeneity, the present work also includes various sensitivity analyses (e.g. intervention category in isolated review, BDI data only, 100% female samples only). These analyses allowed for a check of robustness and generalisability of results by means of mainaining constant various methodological factors (e.g. outcome measure) or sample characteristics (e.g. gender). We found that psychological interventions were robustly associated with significant reductions in comorbid depression relative to control conditions (i.e. across sensitivity analyses), increasing certainty in, and generalisability of, effects. We found most evidence and certainty for TF-CBT, particularly when considering long-term efficacy. Relatedly, the present work also examined mid- and long-term outcome data, without applying restrictions in regard to follow-up periods. As such, the present work was able to synthesise the most long-term outcome data available in the literature and show that reductions in comorbid depression relative to control conditions remain robust across time, which is very informative for clinical practice.

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The present work was also able to perform head-to-head comparisons in the light of a sufficient number of trials directly comparing different types of psychological interventions (e.g. TF-CBT versus EMDR or TF-CBT versus non-trauma-focused interventions). While we found no evidence for significant differences in efficacy between TF-CBT and EMDR, we found some evidence for superior efficacy of TF-CBT relative to non-trauma-focused interventions, which is highly relevant for clinical practice. Lastly, the present work followed gold standard guidelines in meta-analytic conduct (e.g. open access preregistration, strict obedience to PRISMA guidelines). Each step in the process (e.g. systematic literature search, data extractions) was performed independently by at least two of the authors, and discrepancies between coders were dealt with in personal discussions among at least three of the authors, maximising the internal validity of each step of the process.

Limitations

Four limitations need to be noted. First, long-term follow-up data were scarce for all interventions except TF-CBT. More data are needed for these interventions to robustly examine their long-term efficacy. Crucially, a lack of data does not negate efficacy. Second, too few trials ($k = 4$) reported treatment response data concerning depression, limiting the certainty in, and generalisability of, synthesised results. In future trials, treatment success rates (e.g. remission rates, response rates) concerning comorbid depression should be reported alongside continuous data (i.e. means and standard deviations). Such additional analysis of dichotomous outcome data is very important in clinical practice given that clinicians report a strong preference for, and greater clinical utility of, such treatment success rates in percentages.[53] From a clinical practice point of view, the interpretation of standardised mean differences such as Cohen's *d* or Hedges' *g* is less straightforward for both affected people (i.e. patients and significant others) and clinicians. However, dichotomous outcome metrics (i.e. treatment success versus non-success) in the form of treatment success rates in percentages are readily interpretable. As such, treatment success rates may facilitate the informed consent procedure by informing subjects more comprehensibly about the potential merits of a given treatment. Third, and related, the analysis of treatment response involved heterogenous definitions. As more data accumulate, future meta-analytic research will be able to analyse data according to definitions of treatment response. Fourth, some treatment categories (e.g. non-trauma-focused interventions, other trauma-focused interventions and MDTs) and the category of active control conditions are heterogenous clusters. Categorisations were based on the number of available trials. As further trials accumulate, more fine-grained categorisations will become feasible in future syntheses, which will enhance the precision of estimated (differential) effects.

In conclusion, this meta-analysis provides strong evidence for the efficacy of psychological interventions targeting adult PTSD in reducing comorbid depressive symptoms. The majority of available evidence and statistical certainty exist for TF-CBT, which robustly produced significant short-, mid- and long-term efficacy across contexts (e.g. in samples from low- and middle-income countries). Further data for other interventions are needed to investigate the robustness and generalisability of results, particularly with regard to long-term effects. There was some evidence of TF-CBT outperforming non-trauma-focused interventions, and this superiority appeared to be stable across time. The findings provide a positive perspective, suggesting that psychological interventions can significantly reduce comorbid depressive symptoms in traumatised populations suffering from PTSD, thus highlighting the potential benefits of incorporating psychological interventions within health care services.

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Supplementary material

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Data availability

The extracted data, R-codes and forest plots for all analyses are available on the Open Science Framework (OSF; https://osf.io/3sd4x/). This step was taken to maximise the transparency and reproducibility of results. All correspondence should be addressed to the corresponding author; without permission of the corresponding author, data may not be used for purposes other than reproduction of the present results.

Author contributions

T.H.H., A.K. and N.M. conceptualised the meta-analysis. T.H.H. supervised the study. T.H.H., A.S.L., L.H. and A.K. performed some of the literature search and data extraction. T.H.H., A.S.L. and L.H. performed the statistical analyses. T.H.H. wrote the first draft of the manuscript. All authors contributed to, and have approved, the final manuscript.

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Declaration of interest

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