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Review Article

Indicated preventive interventions for depression in children and adolescents: A meta-analysis and meta-regression

Richard Ssegonja[a,*], Camilla Nystrand[a], Inna Feldman[a], Anna Sarkadi[a,c], Sophie Langenskiöld[a,b], Ulf Jonsson[d,e,f]

[a] Department of Public Health and Caring Sciences, Uppsala University, Sweden
[b] Department of Learning, Informatics, Management and Ethics (LIME), Karolinska Institutet, Sweden
[c] Department of Women's and Children's Health, Uppsala University, Sweden
[d] Center of Neurodevelopmental Disorders (KIND), Division of Neuropsychiatry, Department of Women's and Children's Health at Karolinska Institutet, Sweden
[e] Child and Adolescent Psychiatry, BUP-KIND, Center for Psychiatry Research, Stockholm County Council, Stockholm, Sweden
[f] Department of Neuroscience, Child and Adolescent Psychiatry, Uppsala University, Sweden

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ABSTRACT

Depression contributes about 2% to the global burden of disease. A first onset of depressive disorder or subsyndromal depressive symptoms is common in adolescence, indicating that early prevention is a priority. However, trials of preventive interventions for depression in youths show conflicting results. This systematic review and meta-analysis investigated the effectiveness of group-based cognitive behavioral therapy (GB-CBT) as a preventive intervention targeting subsyndromal depression in children and adolescents. In addition, the impact of different covariates (type of comparator and use of booster sessions) was assessed. Relevant articles were identified from previous systematic reviews, and supplemented with an electronic search spanning from 01/09/2014 to 28/02/2018. The retrieved articles were assessed for eligibility and risk of bias. Relevant data were extracted. Intervention effectiveness was pooled using a random-effects model and the impact of covariates assessed using meta-regression. 38 eligible articles (34 trials) were obtained. The analysis showed GB-CBT to significantly reduce the incidence (relative risk 0.43, 95% CI 0.21–0.87) and symptoms (Cohen's d −0.22, 95% CI −0.32 to −0.11) of depression at post-test compared to all controls. Comparisons with passive comparators suggested that the effect decayed over time. However, compared to active controls, a significant intervention effect was evident only after 12 month or more. Our results suggest that the preventive effect of GB-CBT wears off, but still lasts longer than the effect of active comparators. Only a few studies included booster sessions, precluding firm conclusions. Future studies should clarify to what extent maintenance strategies can prolong the preventive effect of GB-CBT.

1. Introduction

Depression is a common disorder with a lifetime prevalence between 10 and 15% (Lepine and Briley, 2011), contributing to 1.84% (1.38%–2.33%) of the total global burden of disease (Disability Adjusted Life Years) (Institute for Health Metrics and Evaluation (IHME), 2016; Whiteford et al., 2013). The disorder is characterized by persistent low mood and loss of interest in previously pleasurable activities (Kasper et al., 2015; American Psychiatric Association, 2013), and is associated with decreased productivity (Lepine and Briley, 2011; Rost et al., 2014), diminished health related quality of life (HRQoL), strains in relationships, poor educational outcomes, unemployment as well as increased utilization of healthcare services (OECD, 2015), and an increased risk of all-cause mortality (Cuijpers et al., 2014), including suicide (Osby et al., 2001; Wulsin et al., 1999).

A significant proportion (over 25%) report that their first episode occurs during adolescence (Kessler et al., 2005), making this a vulnerable time. A substantial proportion of adolescents also have subsyndromal symptoms and go undiagnosed or unmanaged (Bertha and Balazs, 2013; Cameron et al., 2011). Depression with an adolescent onset is associated with an increased risk of depression in adulthood (Jonsson et al., 2011; Copeland et al., 2013). In addition, there is a large

Abbreviations: GB-CBT, group-based cognitive behavioral therapy; SMD, standardized mean differences; MDD, Major Depressive Disorder; EOI, end of intervention; ES, effect size; RR, relative risk

* Corresponding author at: Department of Public Health and Caring Sciences, Uppsala University, Husargatan 3, 751 22 Uppsala, Sweden.
E-mail address: richard.ssegonja@pubcare.uu.se (R. Ssegonja).

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cost burden associated with depressive disorders (Hu, 2006; Gustavsson et al., 2011). In view of the early occurrence of depressive symptoms and the poor prognosis later in life, prevention has received considerable attention (Cuijpers et al., 2012). Specifically, preventive interventions have shown to be less costly than treatment and have a potential to reduce symptom severity (Hetrick et al., 2015; Garber et al., 2016; Crowe and McKay, 2017; Werner-Seidler et al., 2017; Yang et al., 2017), thus being cost-effective (Mihalopoulos and Vos, 2013). This is especially true for indicated preventive interventions for depression in adolescents (Garber, 2006; Rasing et al., 2017). Indicated preventive interventions are preventive measures targeted to people with symptoms that are either too few or not severe enough to warrant a diagnosis of e.g. Major Depressive Disorder (MDD). Indicated preventive interventions for depression in children and adolescents have demonstrated moderate effects, showing relative risks between 0.29 and 0.78 in comparison to all controls (Horowitz and Garber, 2006; Stice et al., 2009; Stockings et al., 2016). There is a range of indicated preventive interventions based on different theoretical grounds e.g. cognitive behavioral therapy, interpersonal therapy, problem solving based therapy, and psychodynamic therapy approaches. Out of these interventions, group-based cognitive behavioral therapy (GB-CBT) is the one that has been most extensively studied. It has been shown to be effective at reducing depressive symptoms when delivered in routine practice and schools (Hetrick et al., 2015; Rasing et al., 2017; Stockings et al., 2016; Bellón et al., 2015; Mendelson and Eaton, 2018). However, some research in the area has found GB-CBT interventions to have inconsistencies, such as low to no effect and that evidence is insufficient to draw generalizable conclusions on effectiveness (Rasing et al., 2017; Merry and Spence, 2007; Brent et al., 2015; Corrieri et al., 2013; Holmes et al., 2018). Most research has also expressed results as continuous outcome measures e.g. Cohen's d, which are not suitable for assessing cost-effectiveness as compared to relative risks. Relative risks can be directly used in health economics modelling to adjust the transition probabilities from one health state to another as opposed to Cohen's d. Furthermore, the effectiveness estimates are regarded the same irrespective of type of comparator (active or passive controls) and presence or absence of booster sessions, which may limit the clinical relevance of the results since earlier research, Gearing et al., demonstrates this difference for booster sessions (Gearing et al., 2013). These distinctions are relevant from both a clinical and economic perspective. Firstly, it is of importance to know if GB-CBT outperforms interventions based on non-specific components related to provision of support and counselling that might be less costly or more practical to implement. Secondly, it is also essential to ascertain if booster sessions have any additional health benefit since its inclusion increases the costs. In addition, to conduct cost-effectiveness evaluations of GB-CBT interventions for children and adolescents, it would require a more definitive evidence synthesis of the effectiveness. Stockings et al. (2016), and Rasing et al. (2017), published the most recent reviews and meta-analyses on the effects of preventive interventions on depression and anxiety in children and adolescents. Stockings' work focused on all types of preventive interventions while Rasing's work focused on targeted (selective and indicated) CBT based interventions but expressed outcomes as continuous outcomes (Cohen's d) rather than RRs. Both studies did not explore the impact of booster sessions and type of comparator (active or passive).

We hypothesized that GB-CBT indicated preventive interventions for depression in children and adolescents would demonstrate to be effective when compared to passive comparators, but that the results would differ for active comparators. We also presumed that inclusion of booster sessions would improve the results.

The aim of this study was to synthesize evidence on GB-CBT *indicated* preventive interventions for depression in children and adolescents with particular focus on:

1) Addressing the inconsistencies in effectiveness of GB-CBT indicated interventions for the prevention of depression in children and adolescents.
2) The effectiveness of GB-CBT indicated preventive interventions in relation to an *active* or *passive* comparator and the impact of booster sessions on intervention effectiveness.
3) Synthesizing and reporting effect sizes in a useful form for inputs in cost-effectiveness assessments and decision-analytic modelling of these interventions.

Addressing the above aims will provide a better base for decision making concerning optimal resource allocation for adoption and implementation of these interventions.

2. Methods

This study was a systematic literature review with a meta-analysis based on the Cochrane Handbook for Systematic Reviews of Interventions (Higgins and Green, 2011), and the Preferred Reporting Items for Systematic Reviews and Meta-analyses (PRISMA) statement guidelines (Moher et al., 2009). This work stems from meta-analyses conducted by Stockings et al. (2016), and Rasing et al. (2017), which were assessed to be of good quality according to the Assessing the Methodological Quality of Systematic Reviews (AMSTAR) framework (Shea et al., 2007).

2.1. Eligibility criteria

Eligibility of the studies considered for inclusion in this work was based on their specified characteristics deemed relevant for answering our research question(s). These study characteristics are summarized in the PICOS below:

P (Population): Children/adolescents aged 12–19 years with depressive symptoms or behaviour indicating depression, not high enough to warrant a diagnosis of a depressive disorder.
I (Intervention): GB-CBT indicated preventive interventions with or without booster sessions.
C (Comparator): Active comparator i.e. other specified preventive treatments for depression or an intervention designed to control for non-specific aspects of treatment for depression (e.g. group counselling and bibliotherapy) and passive comparators e.g. waitlist and treatment as usual (assessment only control with the participants free to seek care).
(Outcome): primary outcome: cases of a depressive disorder (dichotomous). Secondary outcome: depressive symptoms over time (continuous).
S (Study design): Randomized controlled trial (RCT).

2.2. Information sources and literature search

Firstly, all the indicated preventive studies included in Stockings' and Rasling's work were reviewed and only those addressing depression prevention using GB-CBT indicated preventive interventions were selected. Thereafter, a literature search was conducted in electronic databases including PubMed, Web of Science, PsycINFO, Medline and the Cochrane Library of Systematic Reviews using a combination of search terms e.g. adolescents, depression, preventive interventions and cognitive behavioral therapy. A search-strategy is presented in the supplementary material *(eSearch strategy (a))*.

The search was limited to English language, and the period 01/09/2014 to 28/02/2018, to supplement the articles investigating indicated preventive interventions already retrieved in the previous meta-analyses. Furthermore, the reference lists of eligible articles were scanned to obtain potentially missed literature.

2.3. Study selection

The retrieved articles from the two earlier meta-analyses, the additional search in electronic databases and scanning reference lists, were then all screened for title and abstract relevance by two of the authors (RS and CN). Thereafter, the selected articles were read independently by two of the authors (RS and CN) in full text to assess eligibility for inclusion in the systematic review and meta-analysis. All disagreements were resolved by consultation with another author (IF) and consensus. An agreement statistic (Cohen's Kappa) was calculated (Higgins and Green, 2011).

2.4. Data extraction

Information about country, intervention site, participants' age, sample size, outcomes, facilitators, assessment tools, intervention features, comparator (s), summary measures of the effectiveness and study duration were extracted by two of the authors (RS and CN) using an agreed upon extraction tool. Disagreements in the extracted data for example whether a comparator is categorized as active or passive, were resolved through discussions with two other authors (UJ and IF).

2.5. Risk of bias in individual studies

The eligible articles were assessed for bias using The Cochrane Collaboration's tool for assessing risk of bias (Higgins and Green, 2011). The assessment included bias in selection (random sequence generation and allocation concealment), performance, detection, attrition, and reporting. Risk of bias was scored 0–2, i.e. high risk of bias = 0, unclear risk of bias = 1 and low risk of bias = 2. The total scores were used in the meta-regression as an explanatory variable to assess the impact of bias on the intervention effectiveness.

2.6. Risk of bias across studies

The risk of bias across studies was assessed through examining the possibility of publication bias. This was subjectively done using radial and funnel plots of standard errors (on an inverted scale) of the individual studies' intervention effectiveness estimates against the logs of the relative risks or Cohen's d. The distribution of the data points (studies) were then studied on the plot to assess for publication bias.

2.7. Intervention effectiveness

The main outcome measure of intervention effectiveness was the relative risk (RR) of depression (a case of depression was defined as one where a patient was diagnosed with a depressive disorder i.e. MDD or/and dysthymia through a diagnostic interview) because of its suitability for use in cost-effectiveness evaluations. As a secondary outcome measure for the sensitivity analysis, we also collected data expressed as standardized mean differences (SMD) e.g. Cohen's d.

2.8. Meta-analytic approach

The quantitative analysis of the systematic literature review (meta-analysis) used the random effects model (REM) (Borenstein et al., 2009), to estimate the pooled intervention effectiveness. The REM incorporates both the within-study and between-study variance in the weighting process for the studies. The Cochrane's Q statistic and I^2 were estimated as measures of heterogeneity across the included studies. The pooled effectiveness results were estimated with 95% confidence intervals and illustrated using forest-plots.

The calculations were performed using the “metafor” package in R, version 3.4.3.

2.9. Additional analyses

Additionally, we performed a meta-regression (Borenstein et al., 2009) to assess the impact of bias, type of comparator and presence of booster sessions on the intervention effectiveness. The intervention effectiveness was regressed on a set of explanatory variables, as mentioned above, to ascertain to what extent the predictors explain the variation in intervention effectiveness. This analysis also followed a random effects method. We also assessed the impact of an interaction between bias and type of comparator on the intervention effects because participants in the passive comparators were thought to be more likely to seek help outside the study as opposed to those in the active comparators.

Furthermore, the numbers needed to treat (NNT) to prevent one case of depressive disorder was calculated for each time-point.

3. Results

3.1. Study selection

A total of 36 articles, excluding duplicates, were obtained from Stockings' (n = 28) and Rasing's (n = 20) work. An additional 1351 articles were retrieved from the electronic search and 8 articles from the reference lists of the selected literature, thus a total of 1407 articles. Of the 1407 articles, 1275 were eliminated based on the title and abstract irrelevance and an additional 67 papers were duplicates. The remaining 65 articles were read in full text and assessed for eligibility. Twenty seven articles *(eExcluded articles (b))* were ineligible and thus excluded as illustrated in Fig. 1. This left a total of 38 articles (Arnarson and Craighead, 2009; Arnarson and Craighead, 2011; Berry and Hunt, 2009; Hunt et al., 2009; Charkhandeh et al., 2016; Clarke et al., 1995; Duong et al., 2016; Horowitz et al., 2007; Gillham et al., 2006a; Gillham et al., 1995; Gillham et al., 2006b; Gillham et al., 2007; Kosters et al., 2015; Balle and Tortella-Feliu, 2010; Manassis et al., 2010; McCarty et al., 2013; McCarty et al., 2011; Poppelaars et al., 2016; Nobel et al., 2012; Roberts et al., 2003; Rohde et al., 2014; Rohde et al., 2015; Seligman et al., 1999; Seligman et al., 2007; Spence et al., 2003; Sheffield et al., 2006; Stallard et al., 2012; Stice et al., 2007; Stice et al., 2008; Stice et al., 2010; Wijnhoven et al., 2014; Yu and Seligman, 2002; Dobson et al., 2010; Clarke et al., 2001; Kowalenko et al., 2005; Singhal et al., 2014; Woods and Jose, 2012; Hyun et al., 2005) with 49 unique comparisons from 34 trials as summarized in supplementary material *(eSummary of included studies(c))*. The Kappa agreement statistic between CN and RS was 0.84 (see supplementary material, *eAgreement statistic (d)*).

3.2. Study features

Overall, the studies were mainly (45%) carried out in the US. The populations studied were children and adolescents with a mean age of 14 years. The interventions were delivered by a wide range of professionals e.g. psychologists, therapists, counsellors, nurses, teachers and therapists. The average number of participants per group was 8 with an average attendance of 6.5 out of 9 sessions with each session lasting on average 70 min. Delivery of each group intervention required 1–2 facilitators who received supervision weekly. The treatment duration was on average 9 weeks with follow-up times of up to 13 months. The comparator was mostly passive but a few studies compared GB-CBT interventions to active comparators. 8 trials included active comparators designed to control for non-specific aspects of psychological treatment (see supplementary material, *eSummary of active controls (e)*), including a group intervention focusing on stressors associated with adolescent depression (Gillham et al., 2007), an individual or group support program (one educative interview) (Duong et al., 2016; McCarty et al., 2013; Singhal et al., 2014), a supportive-expressive group intervention (Stice et al., 2008; Stice et al., 2010), a group-based

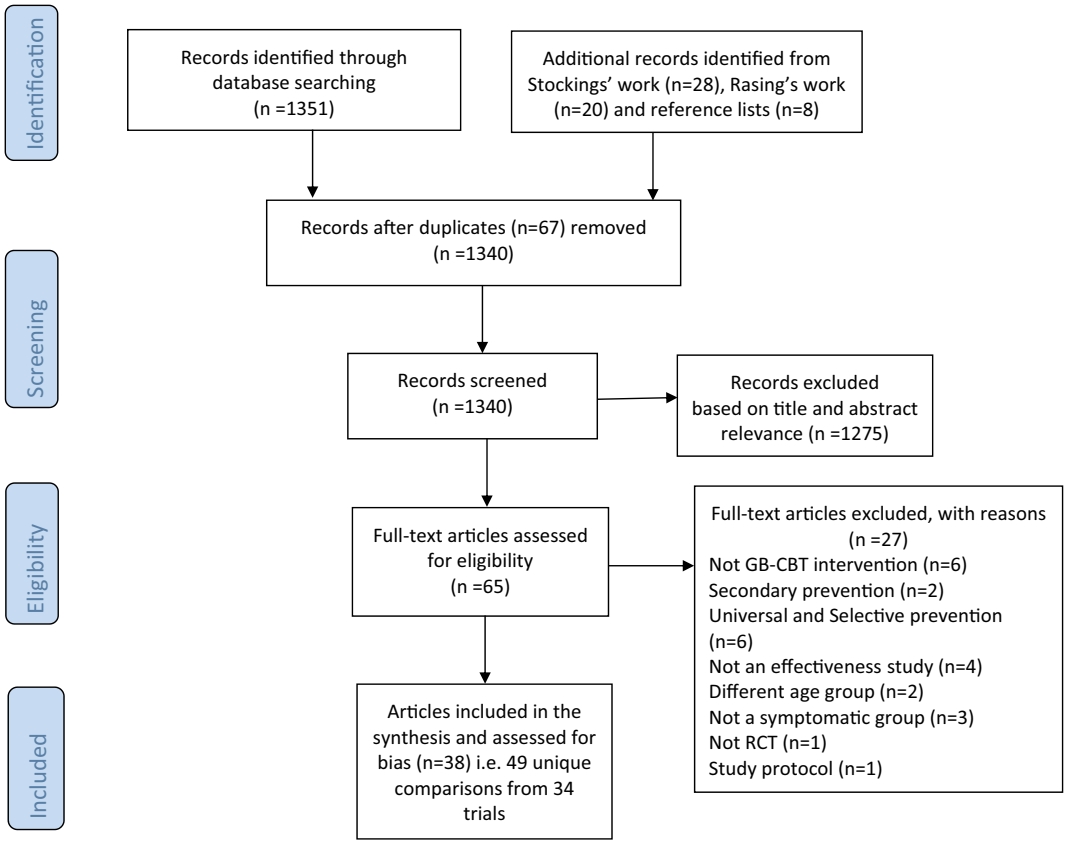


Fig. 1. Flow chart showing the selection of studies.

attention control intervention (Stallard et al., 2012; Dobson et al., 2010), a structured activity group (Nobel et al., 2012), and an expressive writing/journaling intervention (Stice et al., 2007). Three trials included CBT delivered as either bibliotherapy (Stice et al., 2007; Stice et al., 2008; Stice et al., 2010) or as an interactive fantasy game (Poppelaars et al., 2016) as a control condition. Finally, one trial used the complementary treatment Reiki as comparator (Charkhandeh et al., 2016). The interventions were delivered in schools (99%) and health centres (1%). The facilitators received training of a mean duration of 2.65 working days (21.24 h) before delivering the intervention. Supervision to the facilitators of the interventions was done face-to-face 87% of the time, the remaining done via phone calls. Supervision lasted on average one hour per week. Only 16% of the studies had included booster sessions with the interventions. A majority of the studies used the Children's Depression Inventory (CDI) (Kovacs, 1985), Becks Depression Inventory II (BDI II) (Beck et al., 1996), The Centre for Epidemiological studies on Depression tool (CESD) (Radloff, 1977), and the Schedule for Affective disorder and Schizophrenia for School Age Children tool (K-SADS) (Kaufman et al., 1997), to screen, measure depressive symptoms or diagnose cases of depression as shown in *eData extraction (f)* in the supplementary material. In the reviewed studies, a case of depression was defined as a diagnosis of MDD or/and dysthymia using a diagnostic interview, (Stice et al., 2009; Clarke et al., 1995; Duong et al., 2016; Manassis et al., 2010; Rohde et al., 2014; Rohde et al., 2015; Seligman et al., 1999; Spence et al., 2003; Stice et al., 2010) and/or a score on a symptoms scale indicative of severe depression (Stice et al., 2007).

3.3. Risk of bias in individual studies

The most fundamental form of bias was performance bias due to the difficulty of blinding the participants and facilitators given the nature of the interventions. However, other forms of bias, especially selection bias and reporting bias, were unclear, based on the scoring system used in this work as seen in *eBias assessment (g)*.

3.4. Risk of bias across studies

There was some evidence of risk of publication bias *(ePublication bias (h and i))*. The funnel plot including all studies (unique comparisons) displays asymmetry in the studies with small samples sizes and negative outcomes (results showing the intervention as being ineffective).

3.5. Intervention effectiveness

3.5.1. Meta-analysis using the primary outcome measure: cases of depression

Results from the random effects model (REM) are shown in the first part of Table 1 and the forest plots in the supplementary material *(eForest plot cases (j))*. GB-CBT indicated preventive interventions significantly reduced the incidence of depressive disorder at all-time points when including all eligible studies. Intervention effectiveness was noted to decay between EOI and 12 month follow-up, and seems to decay more post the 12 months measurement as seen in Fig. 2. Reducing the sample to studies with a passive comparator maintained the intervention effectiveness at 6 and 12 months after EOI, whereas when comparing to active comparators, the results became non-significant for the first six months, and later significant at twelve months or more.

3.5.2. Meta-analysis using the secondary outcome measure: SMDs

In the second part of Table 1 and supplementary material *(eForest plots Cohen's d (k))*, the results of the random effects model when using SMDs as the outcome measure are shown. GB-CBT indicated preventive interventions significantly reduced symptom severity of depression at all-time points when analyzing the whole sample of studies and when

Table 1
Effectiveness results for the indicated GB-CBT preventive interventions.

		Caseness		SMD		
Time frame	No. of unique comparisons	No. of participants	RR (95% CI)	No. of unique comparisons	No. of participants	d (95% CI)
All interventions						
Post treatment	8 (7 trials)	1461	**0.43 (0.21–0.87)**	43 (33 trials)	7525	**−0.22 (−0.32 to −0.11)**
6 months	13 (7 trials)	1948	**0.6 (0.41–0.87)**	30 (21 trials)	4751	**−0.08 (−0.15–0.00)**
12 months	7 (6 trials)	1246	**0.50 (0.38–0.66)**	21 (17 trials)	4480	**−0.24 (−0.41 to −0.08)**
> 12 months	7 (6 trials)	1311	**0.78 (0.64–0.94)**	12 (9 trials)	1896	**−0.14 (−0.23 to −0.04)**
Active comparators						
Post treatment	4 (4 trials)	695	0.43 (0.17–1.08)	16 (12 trials)	2519	−0.09 (−0.34 to −0.15)
6 months	7 (4 trials)	930	1.01 (0.52–1.95)	11 (7 trials)	1341	0.02 (−0.09–0.12)
12 months	3 (3 trials)	526	**0.50 (0.28–0.90)**	7 (7 trials)	1526	−0.06 (−0.21–0.09)
> 12 months	2 (2 trials)	393	**0.62 (0.39–0.98)**	3 (3 trials)	562	**−0.17 (−0.33 to −0.00)**
Passive comparators						
Post treatment	4 (4 trials)	766	0.35 (0.09–1.33)	27 (26 trials)	5154	**−0.29 (−0.39 to −0.18)**
6 months	6 (6 trials)	1018	**0.41 (0.25–0.68)**	19 (18 trials)	3410	**−0.13 (−0.23 to −0.03)**
12 months	4 (4 trials)	720	**0.49 (0.35–0.69)**	14 (13 trials)	2954	**−0.35 (−0.61 to −0.09)**
> 12 months	5 (5 trials)	918	0.82 (0.66–1.01)	9 (8 trials)	1334	**−0.13 (−0.25 to −0.01)**
Booster sessions						
Post treatment	1 (1 trial)	217	0.95 (0.22–4.12)	7 (6 trials)	2590	−0.21 (−0.45–0.04)
6 months	1 (1 trial)	212	0.98 (0.22–4.26)	4 (4 trials)	1143	−0.08 (−0.28–0.12)
12 months	_	_	_	5 (4 trials)	2050	−0.23 (−0.58–0.12)
> 12 months	2 (2 trials)	338	0.82 (0.62–1.07)	3 (3 trials)	622	−0.02 (−0.18–0.14)
No booster sessions						
Post treatment	7 (6 trials)	1244	**0.33 (0.14–0.75)**	36 (27 trials)	4935	**−0.22 (−0.34 to −0.10)**
6 months	12 (6 trials)	1736	**0.58 (0.38–0.87)**	26 (17 trials)	3608	−0.07 (−0.16–0.01)
12 months	7 (6 trials)	1246	**0.50 (0.38–0.66)**	16 (13 trials)	2430	**−0.25 (−0.45 to −0.05)**
> 12 months	5 (4 trials)	973	**0.74 (0.56–0.97)**	9 (6 trials)	1274	**−0.19 (−0.30 to −0.08)**

Note: Bold highlight: significant results based on significance level (*p*-value) of 5%.
SMD: Standardized Mean Difference.
Cohen's d = Mean difference/Pooled standard deviation.
RR: Relative Risk.

selectively using the sample of studies with passive comparators. In similarity with the outcomes of the first meta-analysis, the meta-analytic results of SMDs using only studies with active comparators yielded significant results past the 12 month follow-up, but not at post-test, 6 and 12 months.

3.6. Additional analyses

3.6.1. Meta-regression model using cases of depression (RR) as the dependent variable

At all time-points, the meta-regression results showed that bias score, nature of the comparator (passive or active) and presence of booster sessions did not significantly impact the intervention effectiveness, illustrated in Table 2.

A model with an interaction term between bias and nature of comparator yielded a non-significant co-efficient implying that the influence of bias did not differ across type of comparator.

3.6.2. Meta-regression model using SMD (Cohen's d) as the dependent variable

The meta-regression results showed that, at 6 months after EOI, holding other factors constant, comparison to an active comparator instead of a passive comparator would significantly decrease the effectiveness of the intervention (Z-value $= -3.19$, $p < 0.01$) as seen in Table 2. Bias was also found to significantly affect the intervention effectiveness negatively (underestimating the true effect) at 6 months (Z-value $= -2.77$, $p < 0.05$). A model with an interaction term for nature of comparator and bias yielded a non-significant co-efficient.

Our results also showed that the NNT to prevent a case of depressive disorder in adolescents with subsyndromal depression at the different time points ranged from nine to forty-two. Comparisons to active comparators yielded higher NNTs at the different time points than the comparisons to passive comparators owing to the low relative risks differences between GB-CBT and the active comparators. See sheet (l) in the supplementary material for values at each time point (eNNT (l)).

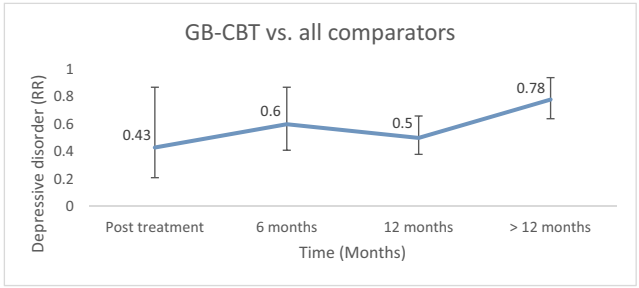
4. Discussion

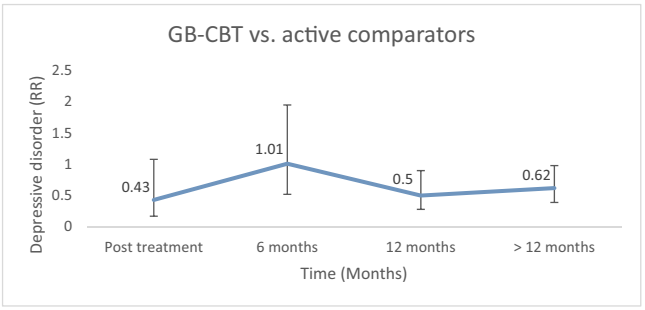
4.1. Summary of evidence

This study aimed to investigate the effectiveness of GB-CBT indicated preventive interventions for depression in children and adolescents. The analyses contribute essentially to existing literature by focusing on the intervention effectiveness when compared to active and/or passive comparators, as well as on the impact of bias and booster sessions on effectiveness. In comparison to all control conditions, there was a reduction in the incidence of depressive disorder(s) at post-test (RR 0.43, 95% CI 0.21–0.87) and an improvement in depression symptoms (d –0.21, 95% CI –0.31 to –0.11).

Thus, GB-CBT indicated preventive interventions seemed to protect the participants from developing a depressive disorder and improve their symptoms. However, a gradual decrease in effects between post-test and 6 months was noticeable. Thereafter the intervention effectiveness increased between the 6 and 12 months before decreasing again at follow-ups > 12 months after EOI. This biphasic pattern could be explained as a delayed effect due to the nature of the intervention since learning and sustaining behavior change takes time. Alternatively, since improvement was noted in both groups over time, it could be the case that the improvement is faster and maintained for a relatively longer period in the treatment groups as opposed to the control groups thus giving the biphasic pattern.

While booster sessions potentially could change the effect over time, our results did not suggest that this had a major impact at any time point. However, these results should be interpreted with caution due to





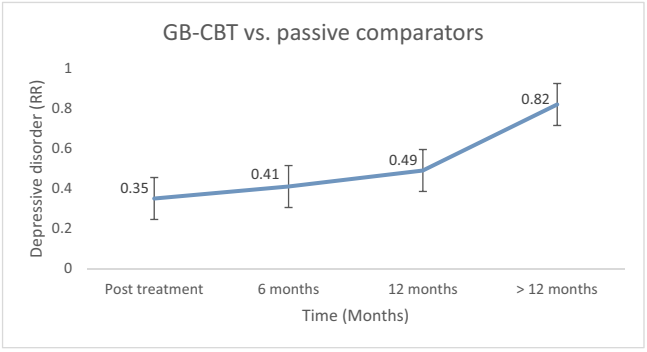


Fig. 2. Decay of treatment effect over time.

Table 2
Meta-regression results.

Covariate	Time-point	Caseness		SMD	
		Z-value	*p*-Value	Z-value	*p*-Value
Bias	Post test	0.789	0.319	−0.355	0.722
	6 months	0.678	0.498	−2.772	**0.010**
	12 months	0.678	0.498	1.131	0.258
	0ver 12 months	0.936	0.349	1.280	0.201
Comparator (passive)	Post test	0.005	0.996	−1.882	0.060
	6 months	−1.568	0.117	−3.189	**0.000**
	12 months	0.06	0.952	−0.729	0.466
	Over 12 months	1.282	0.2	−0.200	0.842
Booster session (yes)	Post test	1.485	0.138	0.538	0.591
	6 months	1.337	0.181	−0.09	0.928
	12 months	_	_	0.195	0.845
	Over 12 months	−0.266	0.79	1.595	0.111

Note: Bold highlight: significant results based on significance level (*p*-value) of 5%.
SMDs: Standardized Mean Difference.
Bias: subjective bias scores assigned to the included studies using the Cochrane handbook for systematic reviews for interventions (Higgins and Green, 2011).

the limited amount of studies with booster sessions and long-term follow-ups included in the analysis.

The separate analyses for passive and active comparators give additional insight into the effect of the intervention over time. The studies using a passive control suggested that the intervention effect decayed steadily. This does not seem to be the result of a fall in study power due to attrition, since the dropout rate was < 30% in the majority of the studies. A more plausible explanation would be that GB-CBT had an effect that was not fully maintained throughout the follow-up period, while passive comparators did not have a meaningful effect at any time. This decay could be due to participants' recidivating back to their previous behavior and ways of thinking. Alternatively it could be due to persistent risk factors that are not addressed with CBT based approaches, such as genetic predisposition, ongoing environmental stressors, or lack of social support.

When comparing GB-CBT to active controls, on the other hand, a significant effect was only evident after 12 months or more. This reversed pattern could imply that the active controls had a temporary effect that was not sustained for > 6 months, after which the sustained effect of GB-CBT was superior to the active comparators. The results of this study generally show that GB-CBT indicated interventions for the prevention of depression in children and adolescents are effective when compared to the control conditions. However, the results after 12 months, where GB-CBT in comparison to active controls seems to be more effective than when comparing to passive controls, is contrary to what would be expected and should be interpreted with caution. This could be due to the vast heterogeneity among the active comparators (ranging from simple expressive writing and bibliotherapy to organized group or therapist sessions) and the small number of trials comparing GB-CBT to an active comparator over the longer term. It could also be that participants on passive comparators are more likely to seek other alternatives in the long term as opposed to those on the active controls.

The most prevalent form of study bias was performance bias which occurred in 70% of the studies. The interventions were group based and were delivered in school settings, which is why it was difficult to blind participants and assessors. This would potentially result into participants in the control group actively seeking alternative help and confirmation bias by assessors. However, in settings of more than one class or school, randomization can be done at a class or school level to overcome this form of bias and control for potential clustering effects in the analysis.

Furthermore, the meta-regression model where the dependent variable was intervention effect measured as Cohen's d showed that at 6 months after the EOI, the nature of the comparator and study bias had a significant impact on the intervention effectiveness. Bias score showed a negative relationship with the intervention effectiveness implying a decreased treatment effect difference between the intervention and control with increased bias in the studies. The same study variables did not have a significant impact on the relative risk estimates. A possible explanation for these findings is the increased power to capture these effect differences since more studies used continuous outcome measures.

4.2. Comparison with other studies

The results from this study are comparable to previous works in this field (Hetrick et al., 2015; Rasing et al., 2017; Stockings et al., 2016). The relative risk of developing depression at post-test (RR 0.43, 95% CI 0.21–0.87) and at 6 months (RR 0.60, 95% CI 0.41–0.87) are comparable to Stockings et al. (Stockings et al., 2016), at post-test (RR 0.48, 95% CI 0.29–0.78) and at 6 months (RR 0.79, 95% CI 0.62–0.99) as well as Rasing et al. (2017), at post-test (d = −0.25; 95% CI –0.38 to –0.12). Unlike Rasing's work, we found that the intervention effectiveness was sustained for more than six months although the first 12 months in a tapering manner and thereafter rapidly decayed. However, when categorising the comparators into active and passive

controls, it yielded, e.g. at post-test, (RR 0.43, 95% CI 0.17–1.08) and (RR 0.35, 95% CI 0.09–1.33) respectively. The distinction between the types of comparator is important for decision-making, as it clearly shows that the magnitude of the effect estimates differ based on the type of comparator. The meta-regression model showed that booster sessions had no significant effect on the intervention effectiveness in both the primary and secondary analyses. This result is different from the findings by Gearing et al. (2013) that showed that CBT interventions with booster sessions outperformed CBT interventions without booster sessions in the management of child and adolescent mood and anxiety disorders. This could be because of the limited number of studies with booster sessions included in our work and the fact that Gearing studied a grouped sample of all mood disorders, looked at both primary and secondary prevention as well as all forms CBT irrespective of mode of delivery which makes it hard to disentangle the observed effects.

4.3. Strengths and limitations

The results of this work are robust due to the restrictive and rather specific eligibility criteria used to select the studies and the different strategies employed to minimize bias. A potential limitation of this study was the difficulty to assess the long term perspective of the intervention effectiveness due to the short follow-up time in a majority of the studies included in this meta-analysis.

Another limitation was that very few studies included in this review had investigated interventions that used booster sessions. This thereby limits any conclusions that could be drawn on the effect of booster sessions on intervention effectiveness.

The limitations of the meta-analytic approach in general as an evidence synthesis method could also be a shortcoming, e.g. the inability to include individual level characteristics in the analysis such as age, ethnicity and social economic status which could influence intervention response. However, the alternative approaches to meta-analysis e.g. integrative- and parallel data analysis could not be performed in our case given that we had no access to individual patient data.

4.4. Implications for clinical practice, policy and research

The results confirm that GB-CBT indicated preventive interventions reduce the incidence and symptoms of depression in adolescents for atleast 12 months or more after EOI, both when compared to passive and to active comparators. Thus, GB-CBT has the potential to prevent or postpone onset of depressive disorder. The potential protective effect of the less comprehensive and less studied interventions used as active comparators (e.g., general support or bibliotherapy) seems to wear off faster than the effect of GB-CBT, which is crucial information to consider when decisions are made about adoption and implementation. However, decisions about implementation are guided by a range of factors. Given the decay of intervention effectiveness over time, the relatively high number needed to treat to prevent a case of depressive disorder, and the limited societal resources, it is essential to establish the cost effectiveness of the intervention. Further, guideline developers should not only consider the intervention's potential to prevent onset of depressive disorder in adolescence, but also the value of delaying onset during this period of rapid cognitive and social development. This work contributes with inputs for efficiency studies in the form of RRs when GB-CBT interventions are compared to both active and passive controls. These results can be used in cost effectiveness evaluations. From a research perspective, the most imminent question seems to be if GB-CBT can be modified to maintain the effect for longer periods of time. Future studies should therefore focus on how and when booster sessions should be provided and if other maintenance strategies embedded in the intervention can improve the outcome over time.

5. Conclusion

Group based CBT indicated preventive interventions for depression in children and adolescents are effective but the effect seems to decay over time. There is a need to further explore the benefit of booster sessions and the appropriate timing for them to be offered. Our results also underscore the value of making a clear distinction between active and passive comparators in order to guide decision-making.

Author contribution

All authors (RS, CN, IF, AS, SL and UJ) were involved in the development of the idea, (RS, CN and IF) did the data collection and analysis, all the authors then worked with the interpretation of results and discussion of this work. They have all agreed to this being the current version of the manuscript.

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Ethical concerns

This study was a systematic literature review using existing published studies and the information from other researchers' work has been well referenced and acknowledged.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ypmed.2018.09.021.

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