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**Late-Life Depression: A Systematic Review of Meta-Analyses and a  
Meta-Analysis of the Effect of Cognitive Behavioural Therapy in  
Older Adults with Co-Morbid Physical Illness**



Presented to the University of Edinburgh in  
Partial Fulfilment of the Requirements for the  
Degree of Doctorate in Clinical Psychology

by

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D. Clin. Psychol. Declaration of own work

*(This sheet must be filled in (each box ticked to show that the condition has been met), signed and dated, and included with all assessments - work will not be marked unless this is done)*

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This thesis is dedicated to our son, Otto Io Craig Huxtable, born 18.6.12

**SECTION 1: Overview of Thesis**

## 1.1. Thesis Overview

This thesis begins with a systematic review (Section 2). The review aims were to identify what meta-analytic studies of psychotherapeutic treatments for late life depression ( $\geq 55$  years) had revealed with regards predictors and moderators of treatment effect. The review is presented in the format required by the journal, Clinical Psychology Review.

Following this, Section 3 outlines the hypothesis for the meta-analysis undertaken in Section 5. Section 4 seeks to place both the preceding systematic review and subsequent meta-analysis in context. It narratively reviews the evidence-base for psychotherapeutic interventions for late-life depression and explores how depression may present differently in late-life: identifying both challenges and protective factors associated with experiences of depression in this age group.

Section 5 presents a meta-analysis of randomised controlled trials evaluating cognitive behavioural therapy for depression in older adults with co-morbid physical illness. This section is also presented in the format required by the journal, Clinical Psychology Review. It was not possible to include full details of the mathematical formulas used to undertake the meta-analysis in the journal article format. Section 6 therefore supplements the methods described in the journal article and facilitates replication of each stage of the meta-analytic process, presenting full details of the formulas and statistical methods employed in the meta-analysis.

The guidelines for submission for Clinical Psychology Review are included in Appendix 1.

## 1.2. Thesis Abstract

**Aims:** To examine the efficacy of CBT for late-life depression in older adults with co-morbid physical illness and to review what has been revealed by meta-analytic studies with regards moderators of treatment in psychological approaches for late-life depression.

**Method:** Systematic literature search and meta-analysis of randomised controlled trials (RCT) evaluating CBT for depression in older adults with co-morbid physical illness and systematic review of meta-analyses examining psychological therapies for late-life depression.

**Results:** Nine papers met inclusion criteria for meta-analysis. CBT was superior to waiting list and treatment as usual control conditions, showing a statistically significant pooled standardised mean difference (SMD) of 0.63 (95 per cent CI, 0.29 to 0.97,  $p = 0.0003$ ). This was largely maintained at follow up (SMD 0.5, 95 per cent CI, 0.08 to 0.92). Sensitivity analysis showed individual CBT yielded a large, statistically significant summary effect size of 0.80 (95 per cent CI, 0.45 to 1.16), but that group CBT did not show statistical superiority over controls. Clinician-rated measures of depression yielded larger effect sizes, with a SMD of 1.57 (95 per cent CI, 0.56 to 2.59,  $p = 0.002$ ) as compared with patient-rated measures: 1.03 (95 per cent CI, 0.75 to 1.31,  $p = 0.0001$ ).

Fourteen meta-analyses met inclusion criteria for systematic review. More recent publication was significantly correlated with increased reporting quality and reduced analysis of moderating factors. Duration of treatment, treatment setting and gender of participants showed no moderating impact on outcome. Depression severity, participant age, treatment modality, and study quality showed no consistent relationship with outcomes. Active or placebo controls were associated with reduced effect sizes when compared with no treatment or waiting list controls. Patient-rated outcome measures were associated with reduced effect sizes as compared with clinician-rated measures.

**Conclusions:** When compared with treatment as usual and waiting list controls Individual CBT is effective in reducing depressive symptoms for depressed older adults with an underlying physical illness. Meta-analytic studies of late-life depression show variable results regarding moderators of treatment efficacy. More high quality studies examining the effectiveness of psychological therapies are needed with clinically representative older populations, particularly, the older-old and those with co-morbid physical illnesses.

**Declaration of interests:** None.



**SECTION 2: Systematic Review (Journal Article)**

**What Meta-analyses have revealed about predictors and moderators of psychological treatments for late-life depression: A systematic review.**

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### Abstract

**Objective:** To systematically review predictors and moderators of treatment efficacy revealed by meta-analytic studies of psychotherapeutic treatments for late life depression ( $\geq 55$  years).

**Methods:** Electronic databases were searched to Feb 2012. Reporting quality and risk of bias were assessed. The relationship between publication date, study quality and number of sub-analyses was explored with regression analyses.

**Results:** Fourteen meta-analyses were included. More recent publication date was correlated with increased reporting quality and reduced analysis of moderating factors. Treatment setting, duration and gender of participants showed no moderating impact on outcome. Physical co-morbidity was associated with reduced treatment efficacy. Depression severity, participant age, treatment modality and study quality showed inconsistent relationships with outcomes. Active or placebo controls were associated with reduced effect sizes compared with waiting list controls. Patient-rated outcome measures were associated with reduced effect sizes compared with clinician-rated measures.

**Conclusions:** Results of moderator analyses in meta-analytic studies of late life depression show inconsistent results. More high quality studies need to be undertaken with clinically representative older populations, in particular, the older old, in order to better understand moderating factors of treatment efficacy in late-life depression.

(Word count: 188 / maximum 200)

Key words: (maximum 6)

PSYCHOTHERAPY, META-ANALYSIS, OLDER ADULTS, DEPRESSION, SYSTEMATIC

## 2.1. Introduction

Depression is the most common psychiatric condition experienced by older people (Beekman, Copeland, & Prince, 1999). It is associated with increased mortality rates (Kane et al., 2010) reduced quality of life (Doraiswamy, Khan, Donahue, & Richard, 2002) and increased morbidity (Penninx, Deeg, & Eijk, 2000). Rates of suicide are higher in this group than in any other age-range (Conwell, Duberstein, & Caine, 2002), and depression is identified as the leading cause of suicide (Baldwin & Wild, 2004). Despite this, depression is under-recognised and under-treated in older people (Karel & Hinrichsen, 2000). Indeed, although the efficacy of psychotherapeutic and other behavioural treatments in treating depressive disorders is well-established (Scogin, Welsh, Hanson, Jamie, & Coates, 2005) older adults are less likely than younger adults to receive adequate or appropriate interventions (Bartels, 2002).

There is increasing recognition that older people should have access to the same range of psychotherapeutic treatments available to younger adults (Department of Health, 2001a; Scottish Executive, 2006; NHS Scotland, 2011). However, making evidence based clinical decisions about which treatment is best suited to an individual older person remains challenging. This is, in part, because identifying specificity of effect in psychotherapeutic outcome studies is far from straightforward. Fiske's (1977, p.24) question, asked at the beginning of the era of psychotherapy meta-analytic research, remains both highly relevant and fiercely contested: "What kind of therapists administering what kind of psychotherapeutic treatments to what kind of patients produce what kind of perceived effects, both immediate and ultimate?"

The question seeks generalised knowledge about what is effective in relation to 'kinds' or classes of therapists, interventions, patients and outcomes. Most studies examining the efficacy of psychotherapeutic interventions have focused on treatment type as the main effect variable. This focus on 'brand name' therapies (Scogin et al., 2005) has occurred despite the fact that non-specific factors such as client hope, therapeutic alliance and the motivation and skill of both therapist and client, have been claimed to explain more of the variance in outcome than treatment approach (Ahn & Wampold, 2001). Meta-analysis has become one tool which can be employed to undertake the task of investigating 'what works for whom?' (Roth & Fonagy, 1996). The promise of meta-analysis, is that it can quantify and indentify potential bias in primary research and provide more accurate generalizations of psychotherapy effects (Matt & Navarro, 1997). In addition, meta-analyses can potentially help us identify those factors which might moderate or predict treatment efficacy.

A moderator is a pre-treatment variable which can be demonstrated to have a statistically significant interaction with an outcome variable (Barber, 2007). If such a relationship can be reliably established, the assessment of moderator variables can help match most appropriate intervention to specific patient needs: a core principle in the stepped care approaches which are now being driven forward by public health organisations (Department of Health, 2009; NHS Scotland, 2011). Meta-analyses frequently undertake analyses of moderating factors: seeking to developing an understanding of under what conditions or for whom a particular treatment should be expected to work (Kraemer, Wilson, Fairburn, & Agras, 2002). However such studies often yield equivocal or contradictory results. This review will systematically identify and evaluate those meta-analyses that have examined psychotherapeutic interventions for depressed older adults and ask to what extent they have helped us answer the question: What works for whom in this group?

### **2.1.1 Rationale for review**

The need for practitioners in real-life clinic settings to draw on the current evidence-base to tailor psychotherapeutic interventions to the older person they are working with is recognised as a key component in best-practice clinical guidelines (National Institute for Health and Clinical Excellence, 2009; Scottish Intercollegiate Guidelines Network, 2010; NHS Scotland, 2011). However, despite a growing body of literature exploring how best to adapt therapeutic approaches to meet the differing needs of depressed older adults (Laidlaw & McAlpine, 2008; Satre, Knight, & David, 2006), an evidence-based understanding of those factors which moderate treatment outcome in late-life depression is not well developed. Although meta-analytic studies are limited in the degree to which they can inform an understanding of specific process issues in therapy, they can support an understanding of the relationship between therapy outcome and key potential moderators of treatment: including therapist variables, treatment modality, and characteristics of the depressed patient. Meta-analyses vary in quality, scope and purpose and although a number of meta-analyses have been undertaken in this area there has been no systematic review of these studies which seeks to critically evaluate and compare their findings with regard to moderators of treatment effect.

### **2.2. Objectives**

This systematic review seeks to answer the following question:

To what extent have meta-analytic studies refined our understanding of what predicts or moderates treatment effects in late-life depression?

The implications of these findings will be discussed within the broader literature which seeks to develop an empirical understanding of the specific factors which moderate psychotherapeutic efficacy.

## 2.3 Methods

### 2.3.1 Eligibility criteria

Eligibility criteria for this review were developed according to methods described in The Cochrane Handbook for Systematic Reviews of Interventions (O'Connor, Green & Higgins 2011).  
Type of study: As the research question in this current study explicitly sought to determine to what extent meta-analytic techniques have helped to understand what might predict and moderate treatment effects, only meta-analytic studies were included. Systematic reviews, narrative reviews and literature summaries were excluded. There was no limit set on date of publication. Only studies in English were included, due to practical restraints of assessors translating other languages. Studies could be published or unpublished. Length of follow up of included studies was not limited.

Participants: Only studies which examined interventions with older adults were included. Older adults were defined as being aged 55+ years. This broad parameter for older age prevented exclusion of potentially relevant studies and facilitated the inclusion of studies which might examine moderating factors related to age. Published research in this area has tended to recruit 'younger-old' participants and adopting this broad age cut off afforded inclusion of earlier relevant meta-analyses.

Population: Only studies including diagnosis of depression, dysthymia or depressive disorders assessed by qualified clinicians or using standardised measures were included. There was no limit on setting (e.g. community, hospital). Meta-analyses which exclusively explored treatment for depression for individuals with specific co-morbid physical conditions were excluded, as the review question sought to identify treatment effects as they might apply to depressed elders in general, and combined interventions taking account of specific co-morbidities were considered unlikely to allow useful generalizability.

Intervention: Meta-analyses examining interventions without an active psychotherapeutic intervention were excluded, for example: exercise therapy; non directive community support, befriending, psychosocial support, behavioural activation. Meta-analyses which included a comparison of psychotherapeutic and pharmacotherapy were also included, but only if data could be usefully extracted from the statistical treatment of psychotherapeutic condition. Comparisons: Studies were included if they involved meta-analytic comparisons between treatment modalities, treatment condition and controls, or undertook meta-analytic analysis of moderating factors.

Outcomes: O'Connor et al. (2011) note that types of outcome should not necessarily be used to exclude studies from meta-analyses, as a variety of measures may be important in understanding efficacy of treatment. However as this review sought to examine treatments of depression, only those which included analyses of standardized rating of depressive symptoms as outcome measures were included.

### **2.3.2. Information sources**

The search to identify studies was conducted in February 2012. The following databases were searched using EBSCOhost: MEDLINE with Full Text, PsycINFO, Psychology and Behavioral Sciences Collection, Health Business Elite, Biomedical Reference Collection: Comprehensive, Library, Information Science & Technology Abstracts, eBook Collection (EBSCOhost), Nursing Reference Center. No limit was set for date of publication. Search terms used were: older adults OR older people OR geriat\* OR elder\* OR late life OR senior; depress\* OR dysthymi\* OR mood; meta-analysis OR systematic review OR quantitative review; psychotherap\* OR psychosocial OR psychological OR cognitive OR behavioural OR psychodynamic OR non-medical OR non-pharmaceutical OR counselling OR inter-personal. Reference lists from included studies were screened to identify any further relevant papers.

First authors of included studies, and The Cochrane Centre for Depression, Anxiety and Neurosis, were contacted to establish whether any unpublished or ongoing meta-analytic studies existed that were relevant to the research question. The World Health Organisation (WHO) International Clinical Trials registry was searched for relevant ongoing studies. To minimise publication bias a search of grey literature was undertaken via System for Information on Grey Literature in Europe (<http://www.opengrey.eu/>); and Web of Science Conference Proceedings Citation Index database. The first author screened abstracts for eligibility. Figure 1 illustrates the search process. The last date searched was the 28.1.12. The full search strategy is included as Appendix 2. Correspondences from contacted authors are included as Appendix 3.

### **2.3.3. Selection**

The author conducted the electronic search and screened first by title, then abstract and finally by reading full texts of journal articles. Studies not meeting eligibility criteria were excluded, with reasons reported for those studies that were excluded at full-text stage.

#### 2.3.4. Data collection process and data items

Data were extracted by the author for the following variables: Number of included studies in the meta-analyses<sup>1</sup>; Number of participants both total and in active intervention groups; Range of number of participants in component studies; Gender ratio across studies; Range of mean age of included studies; Overall mean age for meta-analysis; Diagnostic composition of component studies; Format of included studies; Inclusion criteria of meta-analysis; Number of sessions: range of mean across studies and overall mean; Reporting of random assignment / blinding; Controls of component studies; Quality assessments used and rating co-efficient reported; Outcome measures used in component studies; Reporting of follow up data; Reporting of drop out data; Main findings; Results of any sub-group analyses.

Any missing data were coded as such, but authors were not contacted to supplement or confirm this due to resource constraints of the reviewer. It was assumed that data reported in meta-analyses with regard to primary studies was accurate, and so was not cross-referenced. This was again due to resource constraint. Some of the data-items were reported incompletely or in different form (e.g. ordinal or percentage data). No attempt was made to convert such information and it was included in original form. An exception was when data could easily be collated from tabular results (for example, if total number of participants was not explicitly reported, but could be summed from data reported on individual studies in tabular form). For each study the author collated, in tabular form and summary form, the main results, the results of any sub-analyses, and any analyses of drop-out undertaken (Appendix 4).

#### 2.3.5. Risk of bias in included studies

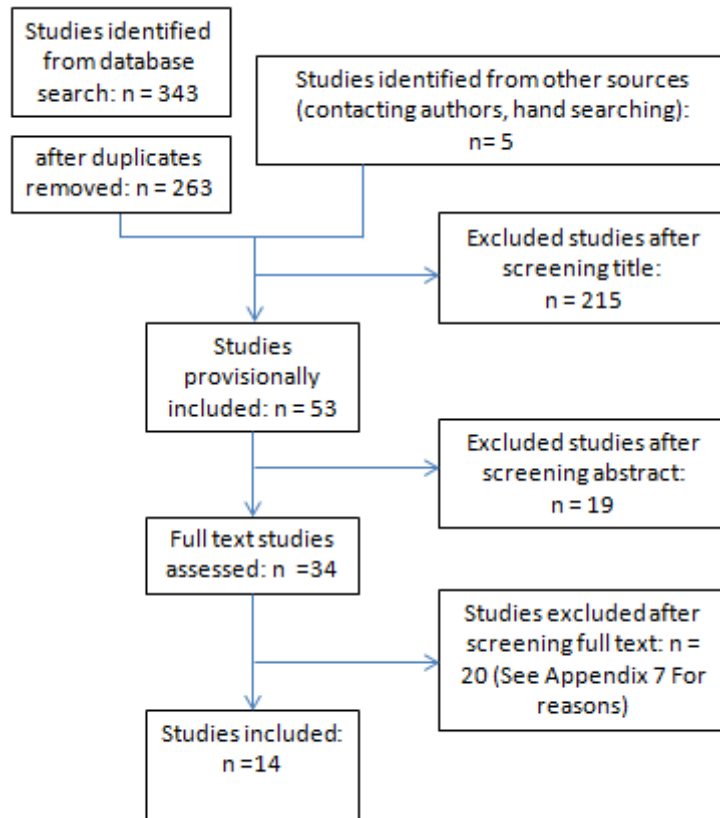
Studies were assessed according to the Preferred Reporting Items for Systematic reviews and Meta-Analyses (PRISMA) guidelines (Moher et al., 2009), which present a 27-item checklist of best-practice for reporting of systematic reviews and meta-analyses and can be used as a framework for the critical appraisal of systematic reviews. The PRISMA guidelines represent a refinement of the previous quality of reporting of meta-analyses (QUOROM) checklist (Moher et al., 1999). The author coded each included meta-analysis against the 27 items set out by the PRISMA checklist (Appendix 6). A score of zero was given if there was no evidence of reporting a particular checklist item. A score of one was given if the study was assessed to only partially meet the reporting guideline for a checklist item. A score of two was given if it was considered a reporting criterion was fully met. Items

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<sup>1</sup> For those trials where only a sub-section of analyses met the eligibility criteria: such as in studies also examining pharmacotherapy, or reporting other outcomes such as life satisfaction, only data for this sub-section of trials was extracted and coded.



five, sixteen, and twenty-three on the checklist (Table 2) were not applicable to all studies so these scores were not included in the comparative reporting quality score for each study. (Full details of scoring given for each study with rationale are included as Appendix 7).



**Figure 1. Flow of studies through the systematic review**

Those items explicitly addressing the reporting of risk of bias (items 12,15,19 and 22 in Table 2) were collated to give an overall score relating to risk of bias. Half of the studies were selected randomly (<http://www.random.org>) and rated according to the PRISMA criteria by an independent assessor (CC). Moher et al. (2009) note the PRISMA checklist is not an instrument designed to quantify the quality of a systematic review, rather a framework to critically appraise quality of reporting. As such, summary scores were not used to rate quality directly, but rather as a heuristic to facilitate assessment of the degree to which studies reported and critically considered the impact of: search and inclusion methodology; sample sizes of included studies; standardisation of psychotherapeutic interventions; expertise of therapists; adherence to therapeutic protocol; randomisation/allocation concealment; intended sub-group analysis; publication bias; risks of bias within meta-analytic methodology.

Consideration of the adequate reporting of sources of bias was used when evaluating and discussing the results of studies. This systematic review did not include in its objectives quantitative synthesis of results, so in line with recommendations outlined by The Cochrane Handbook for Systematic Reviews of Interventions (Higgins & Green, 2011), no formal attempt to weight studies according to quality, or controlling for bias was undertaken.

### 2.3.6. Risk of bias across studies

Risks of bias that may affect cumulative evidence were considered and the degree to which each study contributes to this was considered by coding study characteristics and assessing studies according to the PRISMA checklist (Moher et al., 2009). The impact of risk of bias across studies was examined by undertaking an analysis of the relationship between moderator analysis, study quality and publication date and seeking to critically evaluate results in light of these factors. Risks of bias as a result of the methodology of this review were identified and evaluated as limitations in the discussion.

## 2.4. Results

### 2.4.1. Results of literature search

Following removal of duplicates, the systematic search yielded a total of 269 studies. A total of 254 articles were excluded because they did not meet the inclusion criteria. 215 were excluded after screening the title, 19 after screening the abstracts. Thirty-five full-texts were evaluated and 21 of these excluded. A summary of reasons for exclusion of full text papers is included as Appendix 7.

### 2.4.2. Study characteristics

Characteristics of the included studies are summarised in Table 1.

#### KEY for Table 1 'Characteristics of the included studies Parts 1-4'

ABS = Affect Balance Scale. BDI = Beck Depression Inventory. Bibl. = Bibliotherapy. BSI-D = Brief Symptom Inventory - Depression Scale. BT = Behavioural Therapy. CBT = Cognitive Behaviour Therapy. Clin = Clinical. ClinR = Clinician-rated. CES-D = Center for Epidemiological Studies Depression Scale. CT. = Cognitive Therapy. Dep. = Depression. Diag. = Diagnosis. DSM = Diagnostic and Statistical Manual. Dysth. = Dysthymia. F = Female. GDS = Geriatric Depression Scale. Gr = Group. HADS = Hospital Anxiety and Depression Scale. HDRS = Hamilton Depression Rating Scale. HSCL = Hopkins Symptoms Checklist. ICD-10 = International Classification of Diseases Version 10. IDS = Inventory of Depressive Symptomatology. Ind = Individual. IDD = Inventory to Diagnose Depression. IPT = Interpersonal Psychotherapy. LSI = Life Satisfaction Index. M = Mean. MA = Meta-analysis. MADRS = Montgomery-Asberg Depression Rating Scale. MACL = Mood Adjective Check List. MajD = Major Depression. MinD = Minor Depression. ModDep = Moderate Depression. MMPI-D = Minnesota Multiphasic Personality Inventory Depression Scale. MUMS = Memorial University Mood Scale. MAACL-R = Multiple Adjective Affect Check List Revised. MMPI-D = Minnesota Multiphasic Personality Inventory Depression Scale. NR = Not reported. PGCMS = Philadelphia Geriatric Centre Morale Scale. PST = Problem Solving Therapy. PsychoEd = Psycho-education. PsyD = Psychodynamic Therapy. QRS = Quality Rating Scale. RA = Random Assignment. RCT = Randomised Controlled Trial. Req. = Required. RT = Randomised Trial. RSS = Rosenberg's Self-esteem Scale. RT = Reminiscence Therapy. SCL-20 = 20-item Symptom Checklist. SD = Standard deviation. SevDep = Severe Depression. SubClin = Subclinical. SCID = Structured Clinical Interview for DSM-IV. SWB = Subjective Well-Being. TAU = Treatment as Usual. WL = Waiting List. Zung = Zung Self-Rating Depression Scale.

Table 1: Summary of the characteristics of included studies: Part 1.

Publication	N° of studies	Overall Participants Intervention / Total	Participants (Range within component studies)	Gender ratio	Mean age range studies	Overall mean age	Diagnostic composition of component studies
Gorey & Cryns (1991)	19	809/NR	NR	60.5% F	NR	69.6	NR
Scogin & McElreath (1994)	17	NR/773	16-162	43% F	62-85	70.5	MajD =4. Clin + SubClin =10. SubClin =3.
Engels & Vermey (1997)	17	471/732	4-108	5/17 NR. F>Male but NR by MA	NR	68.6	ModDep = 103. SevDep: 258 (participants)
Cuijpers (1998)	14	634/799	20-162	55%-98% F.	55-83	NR	6/14 Diag. according to DSM/research criteria
Pinquart & Sorensen (2001)	122	NR	10-134 (M 21 in intervention arms)	Mean 71% F	55-87	M = 71.4 (± 7.1)	NR
Bohlmeijer <i>et al.</i> , (2003)	20	959	NR	14/20 > 66% F	NR	M: NR. 9/12: 75+	5/20 = severe. 15/20 = mild-moderate.
Cuijpers <i>et al.</i> , (2006)	25	1937	14-415	NR	NR	≥ 50 = 1 ≥ 55 = 5 ≥ 60 =10 ≥ 65 =7	15/25 = Any diag. of Dep. (9 = MajD.) 10/25 diag. not req.
Pinquart <i>et al.</i> , (2006)	32/89	1407	NR	66% F	NR	70.4	9/32 = MajD
Chin (2007)	6/15	95/178	25-35	85% F	75-82	79.3	Formal diagnosis of depression not required
Pinquart <i>et al.</i> , (2007)	57	1956	NR	67% F	NR	71.77	Self-Rated: 21/75 = MajD. 51/75 =MajD/MinD/Dysth. 3/75 = MinD/Dysth. Clin-Rated: 20/49 =MajD. 25/49 =MajD/MinD/Dysth 3/49 = MinD/Dysth.
Wilson <i>et al.</i> , (2008)	7	NR/153	30-262	3/12 NR. Mean of 9/12: 70% F	NR	NR	Detailed diagnostic composition of each study recorded: Range: Dysth, MinD to MajD.
Peng <i>et al.</i> , (2009)	14	445/705	NR	NR	NR	NR	NR ('depression' key word in search)
Samad <i>et al.</i> , (2011)	4	186/256	30-95	38% - 85% F	66-68	NR	2/4 = MajID. 1/4 = HDRS≥10. 1/4 = HDRS≥10, BDI ≥10, GDS ≥11.
Krishna <i>et al.</i> , (2011)	6	NR	<30 - 238	96%, 75%, 80%, 75%, 50% F One study: NR	66 -84	NR	5/6 Diag. according to DSM/research criteria. 2/6 excluded severe depression. 6/6 'mild-moderate' depression.

Table 1: Summary of the characteristics of included studies: Part 2.

Publication	Outcome measures	Follow up	Drop out
Gorey & Cryns (1991)	BDI, HDRS, Zung, account for 75 % of measures used.	4/19 although details not given	23.10%
Scogin & McElreath (1994)	Coded 'self' (e.g., BDI) or 'clinician' (e.g. HDRS)	NR	NR
Engels & Vermey (1997)	HDRS =4. BDI =4. BDI-short =3. GDS =3.	8/17, 1month-1yr. Effect sizes maintained.	4/17 studies no info given. Mean of others 16%. 5 studies reported 0% drop-out.
Cuijpers (1998)	BDI, HDRS, GDS, MMPI-D, Zung, BSI-D. Breakdown not reported.	6/14 = 1month-2yr.	2/14 did not report. For others: dropout rate: 0.9-0.47 (mean 0.23 , SD 0.13.)
Pinquart & Sorensen (2001)	Self-rated Dep. = 57/122. Clin rated =12/122. Self-rated SWB = 84. BDI=22. GDS=11. Zung =8. CES-D =5. HDRS (7) LSI(18)RSS (12) PGCMS (8) ABS (5)	Most studies with follow up didn't use control groups. No sig difference at follow up in over 50% of analyses, but limited data.	NR
Bohlmeijer <i>et al.</i> , (2003)	BDI. HDRS, GDS MACL, Zung, MADRS, MUMS, MAACL-R. Breakdown not reported.	NR	5/20 NR. 4 studies dropout..25%. Mean dropout across studies 16%.
Cuijpers <i>et al.</i> , (2006)	HDRS, SCID, BDI, GDS, HSCL-20, MMPI-d, Zung, CES-D, HADS-D, IDS, HDRS, MADRS, IDD. Breakdown not reported.	2/25 studies included data comparing follow up & control. 4/25 allowed calculation of post-test/follow up, (range 3months -1 yr). No analysis reached significance.	10 studies = <20%. 8 studies = 20-30%. 5 studies= >30%. Two studies = NR.
Pinquart <i>et al.</i> , (2006)	For all studies (including drug trials) HDRS =61. MADRS=4. BDI=21. GDS =20. Other =29.	NR	NR
Chin (2007)	GDS =5. BDI =1.	NR	Three studies 0%. mean of others: 12%
Pinquart <i>et al.</i> , (2007)	GDS =22. BDI =19. HDRS =17. CESD =6. Zung =6. Clinical Interview =5. Other 'validated measures' =11.	21% reported follow up	18.9% of intervention participants and 18% of control groups (from 50 samples)
Wilson <i>et al.</i> , (2008)	HDRS =8. GDS =7.	One included study was a follow-up trial. Another recorded follow up between 12-16 weeks.	Drop out coded and reported for 9/12. 2/12 dropout unclear. 1/12 not reported. High dropout rates across trials. Difference between therapy and control rates reported.
Peng <i>et al.</i> , (2009)	Including' SCL-20, HDRS, BDI, GDS. Breakdown not reported.	607/705 received 'follow up' unclear if this actually means 'study completers'	Drop-out rates not reported: analysis undertaken: those receiving treatment for depression with or without psychotherapy( 5 studies)
Samad <i>et al.</i> , (2011)	BDI =3. Zung =1.HDRS =4. GDS=3. BSI=1.	3-month follow up (one study) considered comparable to immediate outcome data for analysis	Reported in all four. Pooled odds ratio was 1.50 (95% CI 0.32–6.96) with no significant difference between trials.
Krishna <i>et al.</i> , (2011)	(HDRS) + (GDS) + (BDI) = 2/6. IDS =1/6 .MADRS = 1/6. HDRS =1/6.	2/6 + no follow up. 3/6 = one yr. 1/6 = 9 months. mean 11.3 months (SD 1.5)	1/6 unclear. 5/6 data detailed.

Table 1: Summary of the characteristics of included studies: Part 3.

Publication	Intervention Conditions.	Controls of Component Studies.	Quality Assessment Used.	Random assignment /Blinding
Gorey & Cryns (1991)	Cognitive-Behavioural 'Psychodynamic' 'Other'. Numbers NR.	NR	NR	NR
Scogin & McElreath (1994)	CBT =7. BT =4. IPT/PsyD =3. RT =8. Eclectic therapy =1.	All studies used control/comparison group: No treatment = 17. Attention placebo =4. Pill placebo = 2. Supportive contact = 1. Comparator intervention =10.	Suydam (1968) Nine dimensions. Two raters: (r) 0.73.	NR
Engels & Vermeij (1997)	CT =7. BT=4. PsyD=4. CBT =5. 'Other' =7.	No control =6. Artificial control calculated to allow synthesis of data. Actual controls categorized: 'Wait list/minimum support' and 'placebo therapy'.	No measure. 5/17 'poor quality': failure to report drop-out or randomisation.	RA =10. Adjusted randomisation =3 Clients matched =2. No info. =3
Cuijpers (1998)	CBT=9. PST =1. BT=1. PsyD =4. Bibl-CBT =2. Bibl-BT =1.	Control = 8, of these waiting list =6.	NR	RA = 12. Method NR.
Pinquart & Sorensen (2001)	Breakdown NR.	No treatment = 96. Attention placebo =38.	'3-point scale.'	NR, but coded in quality assessment
Bohlmeijer <i>et al.</i> , (2003)	15/20 = RT. 7/20 = life review.	No treatment =13. Psych placebo =9. WL =1. RT =3	4/20 Assessed as high quality	RA =15 Method NR.
Cuijpers <i>et al.</i> , (2006)	CBT = 12. BT=4. RT =4. IPT =4. PST =4. PsyD=2 'Bibl =4. CT =1.	Control =17. WL =8. TAU =4. Placebo =3. Other = 3. Comparison between treatments =8.	Higgins & Green (2005)	Independent RA =2. 13/25 blinding of assessors
Pinquart <i>et al.</i> , (2006)	Coded 'CBT' or 'other'. Breakdown NR.	Drug placebo =4. Attention placebo =6. TAU =2. WL=22.	Juni <i>et al.</i> , (2001)	RA =29 Method NR.
Chin (2007)	RT.	All studies used control: type not recorded.	Chalmers <i>et al.</i> , 1990.	RA =4. Method NR.
Pinquart <i>et al.</i> , (2007)	CBT =13. BT =11. CT =10. RT =8. PsychED =8. PsyD= 3. IPT =3. 'Other' =25.	Coded 'active placebo' or 'other' data NR.	Four criteria: random assignment; >10 participants in each group; equivalence of control group; exact effects reported.	RA =87.9% Method NR.
Wilson <i>et al.</i> , (2008)	CT, BT, CBT, PsyD, Bilbl. Detailed, but not collated.	All studies used control: types detailed, not collated.	QRS (Moncrieff, 2001)	RA =9 Details of blinding reported.
Peng <i>et al.</i> , (2009)	CBT, n = 138. RT, n = 109. 'psychotherapy' n = 100.	All studies used control: type not recorded.	NR	NR
Samad <i>et al.</i> , (2011)	BT =4.	Delayed treatment =3.	Higgins and Green (2008) Cochrane collaboration risk of bias tool.	RA= 4. Method NR. Allocation, concealment or blinding not clear
Krishna <i>et al.</i> , (2011)	CBT (all studies)= 6. Comparators: RT=2 Other =4.	All studies used control/comparator. RT=2. Group visual imagery =1. Education =1. WL=3. Educational group =2.	QRS (Moncrieff <i>et al.</i> , 2006) inter-rater reliability of k 0.76 (95%CI: 0.48, 1.07).	RA=6. Method NR.

Table 1: Summary of the characteristics of included studies: Part 4.

Publication	Format	Inclusion Criteria	Sessions (range)	Sessions (mean)
Gorey & Cryns (1991)	Gr = 19.	Not systematic: 'relevance to question'; Inclusion of data to allow MA. Although ≥65yrs detailed in abstract, 27% of participants between 55 yrs and 64 yrs.	10-160	56.5
Scogin & McElreath (1994)	Gr = 10. Ind =2. Self-directed =2.	≥60 yr; Psychosocial treatment for depression; control comparison; standardized depression outcome measure.	5-46	12
Engels & Vermey (1997)	Gr =9. Ind =18. Ind and Group =1.	No specific age criteria: 'elderly'. Evidence of 'depressive complaints'; Inclusion of data to allow MA.	4 - 20	NR
Cuijpers (1998)	Gr =6. ind =6. Bibl =2.	≥55 yrs. Psychological intervention for depression; Active recruitment; pre/post test data.	5-46	'most' = 10-20
Pinquart & Sorensen (2001)	Gr =65.4%. Ind =27.7%. (Gr+Ind) + NR =6.9%	≥ 55 yrs. Psychosocial or psychotherapeutic intervention compared to control; outcome measure of depression or well-being; inclusion of data to allow MA.	1-250	Median = 9
Bohlmeijer <i>et al.</i> , (2003)	Gr =14. Ind =6.	No age inclusion criteria. Reminiscence/life review; Depression outcome measure; control or comparison group.	9/20 = <6. 9/20= 7-12. 2/20= >7.	NR
Cuijpers <i>et al.</i> , (2006)	Gr =7. Ind =14. Bibl =4.	≥50 yrs. RCT comparing psychological treatment to control or other treatment; Clinically relevant depressive symptoms; Inclusion of data to allow MA.	4-20	NR
Pinquart <i>et al.</i> , (2006)	Gr (inpatient) = 9. Others NR.	≥ 60; Diagnosis of depressive disorder; control used; Inclusion of data to allow MA.	NR	9.4 weeks
Chin (2007)	Gr =6.	≥50 yrs. Reminiscence; controlled trial; before 2001; life satisfaction, happiness, self-esteem or depression outcomes; Pre-test/post-test; >5 in each group.	6(2hr) -16 (1hr)	10.3
Pinquart <i>et al.</i> , (2007)	Gr =61.7%	≥60 yrs; MajD/MinD or Dysth. (ICD-10, DSM-IV); psychological or behavioural intervention compared with control; sufficient data to estimate depression change score.	NR	15.2
Wilson <i>et al.</i> , (2008)	Gr =4. Ind =5. Bibl =3.	≥55 yrs. RCT or cluster RT ;diagnosis of depression using diagnostic criteria or standardized rating scale;	6-20 (weeks following randomisation)	NR
Peng <i>et al.</i> , (2009)	NR	≥55 yrs. RCT; drop-out rates > 50%; 'depression' although not defined.	NR	NR
Samad <i>et al.</i> , (2011)	Ind =3. Bibl =1.	≥55 yrs. Behaviour Therapy; RCT; diagnosis of depression using standardized outcome measure/diagnostic criteria.	10 - 20 (excl Bibl)	14.6
Krishna <i>et al.</i> , (2011)	Gr =6.	≥50 yrs. RCT or cluster RT; formalised psychotherapeutic treatment; diagnosis of depression using standardized outcome measure/diagnostic criteria; at least one group; (>3) in group.	8 =1. 10 =1. 11 =1. 12= 2. 24 =1. (weeks)	12.8

### 2.4.3. Risk of bias within studies.

There was an 89% agreement between raters with a Kappa of 0.82 (95% CI 0.75 to 0.89) (Appendix 8). Overall, the quality of reporting was not optimum, meaning it was often not possible to ascertain actual methodology and therefore adequately account for potential sources of bias within studies.

Search and inclusion methodology was particularly poorly reported across studies. Only Wilson, Mottram and Vassilas (2008) reported in such a way that would facilitate a fully systematic and replication of the process. One potential source of bias in Wilson et al.'s (2008) search and inclusion criteria was the exclusion of potentially eligible studies that were in the process of review. However, transparent reporting of this facilitated potential further analysis as to whether these named studies may have altered findings. Only four studies were judged to report on risk of bias adequately. Three of these studies (Krishna et al. 2011; Samad, Brealey & Gilbody, 2011; Wilson et al., 2008) reported on the insufficiency of randomisation/allocation concealment. Other studies either failed to report on randomisation or reported this variable without critical appraisal of its impact on bias across or within studies. Wilson et al. (2008) reported on whether studies had evidenced therapist experience, adherence to therapeutic protocol and whether studies had evidenced standardisation of psychotherapeutic interventions. These factors were not considered systematically in other included reviews.

All meta-analyses included primary studies with small sample sizes, but not all studies identified this as a source of potential bias or considered this in their conclusions. Reporting of intended sub-group analysis, and evaluation of risks of bias within meta-analytic methodology were not routinely reported in the majority of reviews. Coding for intention to treat analysis was only reported by Pinguart, Duberstein and Lyness (2006) and Wilson et al. (2008).

**Table 2: Assessment of reporting quality. The scores allocated for each PRISMA checklist criteria 1-27 are tabulated. Detailed PRISMA criteria outlined in Moher et al., (2009). '0' = Did not meet criteria. '1' = Partially met criteria. '2' = Fully met criteria. Mean scores for each criterion across studies is listed in far right column. Items 5, 16, and 23 were not applicable to all studies and were therefore not included in overall 'Quality of reporting Score' (maximum value = 48). Reporting of risk of bias sums items 12, 15 19, 22 (maximum value = 8).**



	Gorey & Cryns (1991)	Scogin & McElreath, (1994)	Engels & Vermey (1997)	Cuijpers, (1998)	Pinquart & Sorensen (2001)	Bohlmeijer et al., (2003)	Cuijpers et al., (2006)	Pinquart et al., (2006)	Chin (2007)	Pinquart et al., (2007)	Wilson et al., (2008)	Peng et al., (2009)	Samad et al., (2011)	Krishna et al., (2011)	Mean score across studies	
<b>Title</b>																
1	Title	2	1	1	2	2	2	2	2	2	2	2	2	2	2	1.9
<b>Abstract</b>																
2	Structured summary	1	1	1	1	1	1	2	2	1	2	1	2	1	1.3	
<b>Introduction</b>																
3	Rationale	1	2	2	2	2	2	2	2	2	2	0	2	2	1.8	
4	Objectives	1	1	1	1	2	1	2	2	1	2	1	0	2	1	1.3
<b>Methods</b>																
5	Protocol and registration	0	0	0	0	0	0	0	0	0	1	0	0	0	0.1	
6	Eligibility criteria	0	1	1	1	1	2	2	1	1	1	2	0	2	2	1.2
7	Information sources	1	1	1	1	1	1	1	1	1	2	1	1	2	1.1	
8	Search	0	0	0	0	0	0	0	0	0	2	0	0	0	0.1	
9	Study selection	0	1	1	1	0	1	1	1	2	0	2	1	2	1	1.0
10	Data collection process	0	1	1	0	0	0	1	2	1	2	1	2	1	0.9	
11	Data items	0	2	2	2	2	1	2	2	2	2	1	2	0	1.6	
12	Risk of bias within studies	1	1	0	0	1	0	0	1	1	1	2	0	2	2	0.9
13	Summary measures	1	2	2	2	2	2	2	2	1	2	2	0	2	2	1.7
14	Synthesis of results	1	2	1	2	2	2	2	2	2	2	0	2	1	1.6	
15	Risk of bias across studies	0	1	0	1	0	1	0	1	2	0	2	0	2	2	0.9
16	Additional analyses	1	0	0	2	1	0	1	0	0	1	2	0	0	1	0.6
<b>Results</b>																
17	Study selection	1	0	1	0	0	2	0	1	0	1	1	2	2	0.8	
18	Study characteristics	0	1	0	2	0	2	2	0	2	2	0	2	2	1.2	
19	Risk of bias within studies	0	1	1	0	0	1	0	1	0	2	0	2	2	0.8	
20	Results of individual studies	0	1	0	1	0	0	2	0	2	1	2	0	2	2	0.9
21	Synthesis of results	1	1	1	2	2	2	2	2	1	2	2	1	2	1	1.6
22	Risk of bias across studies	0	0	1	0	1	0	0	2	1	1	0	1	1	0.6	
23	Additional analysis	1	2	2	2	2	2	2	2	0	2	2	1	0	0	1.4
<b>Discussion</b>																
24	Summary of evidence	1	1	1	1	2	2	2	2	1	1	2	1	2	2	1.5
25	Limitations	0	1	2	1	2	1	2	2	2	1	2	1	2	1	1.4
26	Conclusions	1	2	2	2	2	2	2	2	1	2	2	0	2	2	1.7
<b>Funding</b>																
27	Funding	0	0	1	0	0	0	2	0	0	2	0	2	2	0.6	
<b>Quality of Reporting Score</b>																
Reporting of risk of bias																
Quality of Reporting Score																
Reporting of risk of bias																
Mean score across studies																



#### **2.4.4. Risk of bias across studies**

In addition to the failure to report or critically evaluate adherence to therapeutic protocols in primary studies, detailed definition of therapeutic categories defined within meta-analyses was frequently not adequately outlined, or linked with primary studies. Thus there was a risk across studies that interventions classed as, for example, 'cognitive' in one analysis, may be grouped within the 'cognitive-behavioural' category in another analysis without this being easy to explicitly cross-reference or check. A significant number of included studies did not adequately report on outcome measures, drop out, control conditions or random assignment, and therefore did not critically appraise the possible risk of bias emerging across studies from synthesizing data without attempting to quantify or consider these factors.

Significant heterogeneity was found in a large proportion of the included meta-analyses and meta-analytic techniques were frequently not adequately reported. There is therefore a significant risk that discrepant results in moderator analysis may be an artefact of methodological approaches. Pinquart and Sorensen (2001), for example, note that the fact that they did not find significant age differences in treatment effects where Engels and Vermey (1997) report a tentative finding in this direction, may be due to the latter authors employing a fixed, rather than random-effects analysis, and thus potentially over-estimating the statistical significance of their results. Many of the included meta-analyses included outcome studies with less than ideal quality and failed to adequately discriminate between poor and good quality studies when reporting data synthesis, making the reliability and validity of comparative analyses difficult to ascertain.

The impact of publication bias was not represented by funnel plots in any of the included studies and generally poorly reported, leading to a high risk of over-estimation of effect sizes across the included studies (Cuijpers, Smit, Bohlmeijer, Hollon, & Andersson, 2010). Two studies (Krishna et al., 2011; Samad et al., 2011) reported that they had insufficient studies to undertake sensitivity and funnel plot analyses. Wilson et al. (2008) reported use of funnel plots, but did not report data. The study by Chin (2007) scored highly for its reporting of bias despite including studies of poor quality, as limitations related to poor quality were clearly delineated and the consequent limited capacity for robust conclusions detailed.

#### **2.4.5. Relationship between date of publication, reporting quality and number of analyses of predictor/moderating factors.**

Earlier studies tended to be reported less systematically and include a greater number of sub-analyses. Linear regression analysis was undertaken using the 'R' statistical and programming

environment (R Development Core Team, 2011). More recent publication date was significantly correlated with increased reporting quality ('A':  $r^2 = 0.33$ ,  $F = 6.004$   $p = 0.03$ ) and reduced analysis of predictors of outcome/moderating factors ('B':  $r^2 = 0.42$ ,  $F = 8.601$   $p = 0.013$ ). Reporting quality and number of analyses were not significantly correlated due to the disproportionate influence of a single outlier (Peng et al., 2009) as revealed by a Cook's distance  $\geq 1$  (Crawley, 2009 p. 401) Excluding the paper by Peng et al., (2009) revealed a significant inverse relationship between reporting quality and number of analyses of moderating factors undertaken ('C':  $r^2 = 0.54$ ,  $F = 13.03$   $p = 0.004$ ). Plots used to validate the model and check for violation of assumptions of linear regression analysis are included as Appendix 9.

#### **2.4.6. Moderators of outcomes in the psychotherapeutic treatment of depression: results from the included meta-analyses.**

As seen in Table 3, treatment moderators explored by the included studies were: severity of depression; modality of therapy; duration of treatment; treatment setting; participant age; gender; presence of co-morbidities; therapist experience; social support. Other factors for which relationship to outcome was explored included: type of therapy; outcome measures used; type of control; study quality.

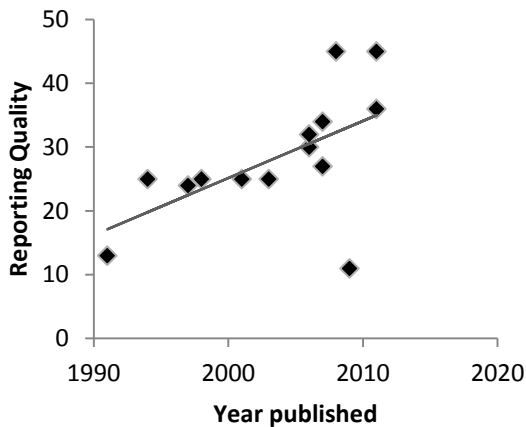
##### **2.4.6.1. Factors that consistently showed a moderating effect on treatment response**

###### **2.4.6.1.1. Presence of co-morbidities (two analyses)**

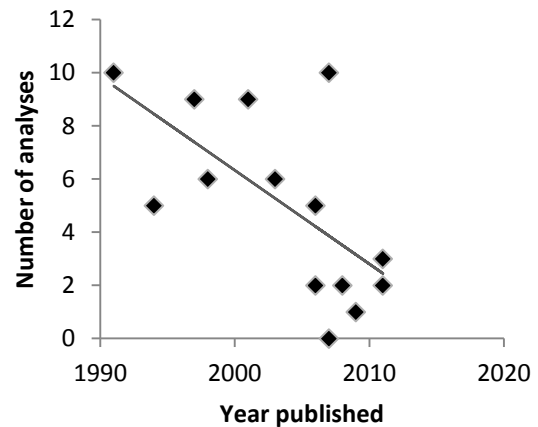
Presence of co-morbidities showed a consistent moderating effect on treatment response, although this was only across two analyses. Engels and Vermey (1997) found that patients classed as having 'multiple complaints' responded less well to treatment ( $d = 0.14$ ) than those diagnosed with either major depression ( $d = 0.86$ ) and other depression diagnoses ( $d = 0.68$ ) and inferred that co-morbidity may inhibit response to therapeutic treatment. These results need to be interpreted with caution as non-psychological interventions were included in the analysis and the natures of secondary diagnoses were not detailed. Pinquart et al. (2007) found significantly weaker improvements of depressive symptoms were found in studies that included participants with physical co-morbidities ( $B = -0.35$ ,  $\beta = -0.22$ ,  $t = -2.23$ ).

**Figure 2. A: Relationship between reporting quality (as determined by PRISMA checklist score) and year of publication. B: Relationship between number of analyses undertaken of potential moderating factors and year of publication. C: Relationship between number of analyses undertaken of potential moderating factors and reporting quality (Dotted line indicates non-significant correlation before removing outlier. Outlier =triangular point).**

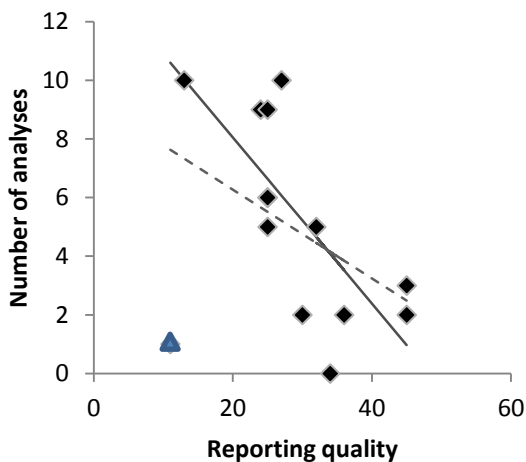
A:  $r^2 = 0.33$ ,  $F = 6.004$   $p = 0.03$



B:  $r^2 = 0.42$ ,  $F = 8.601$   $p = 0.013$



C:  $r^2 = 0.54$ ,  $F = 13.03$   $p = 0.004$



## 2.4.6.2. Factors that consistently showed no moderating effect on treatment response

### 2.4.6.2.1. Duration of treatment (six analyses)

Gorey and Cryns (1991) reported non-significance without supporting analyses or data. Scogin and McElreath (1994) report a non-significant association of effect size with number of treatment sessions ( $ES = -0.20$ ,  $n = 12$ ). Engels and Vermey (1997) included artificial controls in their analysis, but when these data were omitted the number of treatment hours was not significantly associated with effect size ( $r = 0.06$ ). Non-psychological interventions were included in this analysis.

**Table 3: Studies arranged according to PRISMA checklist reporting quality score. Ticks indicate factors reported as significantly associated with treatment outcome. Crosses indicate factors reported as not significantly associated with treatment outcome. A dash indicates that this factor was not analysed. If a study undertook analysis of drop-out this is indicated with a tick. Total number of analyses of potential moderating factors is listed in the final column.**

	PRISMA Checklist Score	Type of Therapy	Severity of depression	Outcome Measure	Modality	Duration	Type of Control	Study Quality	Setting	Age	Gender	Co-morbidities	Size of Group	Therapist Experience	Social Support	Drop-out analysis?	N o of analyses
Peng et al., (2009)	12	✗	-	-	-	-	-	-	-	-	-	-	-	-	-	✓	1
Gorey & Cryns (1991)	13	✗	✓	-	-	✗	✓	✓	✗	✗	✗	-	✓	-	✓	✓	10
Engels & Vermey (1997)	24	✓	✗	✓	✓	✗	✓	-	-	✓	✗	✓	-	-	-	-	9
Scogin & McElreath (1994)	25	✗	✗	-	✗	✗	-	✗	-	-	-	-	-	-	-	-	5
Cuijpers (1998)	25	✗	✗	-	✗	✗	-	-	-	✗	✗	-	-	-	-	✓	6
Pinquart & Sorensen (2001)	25	✓	✓	✓	✓	✗	✗	✗	✗	-	-	-	-	✓	-	-	9
Bohlmeijer et al., (2003)	25	✗	✓	-	✗	-	-	✗	✗	-	✗	-	-	-	-	-	6
Pinquart et al., (2007)	27	✓	✓	✗	✗	✗	✓	✓	✗	✗	-	✓	-	-	-	✓	10
Pinquart et al., (2006)	30	✓	-	✓	-	-	-	-	-	-	-	-	-	-	-	-	2
Cuijpers et al., (2006)	32		✗	✗	✗		✗	-	-	-	-	-	-	-	-	-	5
Chin (2007)	34	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	0
Krishna et al., (2011)	36	-	-	✓	-	-	✓	-	-	-	-	-	-	-	-	-	2
Samad et al., (2011)	44	✗	✓	✓												✓	3
Wilson et al., (2008)	45	✗	-	✓	-	-	-	-	-	-	-	-	-	-	-	✓	2
Effect found		4	5	6	2	0	4	2	0	1	0	2	1	1	1		
No effect found		8	4	2	5	6	2	3	4	3	4	0	0	0	0		

Cuijpers (1998) reported that regression analysis yielded no correlation between number of treatment sessions and effect size, although data are not reported. Pinquart and Sorensen (2001) compared psychotherapeutic interventions with more than nine sessions with those with nine or

fewer sessions. Outcomes were not significantly different on either self-report (sr) or clinician rated (cr) measures. For interventions with more than 9 sessions,  $ES(sr) = 0.59$ ,  $ES(cl) = 1.21$ . For those with less than nine sessions,  $ES(sr) = 0.40$ ,  $ES(cl) = 0.76$ . Piquart et al. (2007) found no significant correlation between number of sessions and outcome ( $B = -0.01$ ,  $\beta = -0.12$ ,  $t = 1.17$ ).

#### **2.4.6.2.2. Treatment setting (four analyses)**

Gorey and Cryns (1991) report non-significance without reporting results of their analysis. Piquart and Sorensen (2001) found mean effect sizes did not differ significantly between community ( $g = 0.51$ ) and nursing home ( $g = 0.39$ ) settings. Bohlmeijer et al. (2003) found that effect sizes for interventions in community and non-community settings overlapped at the 95% confidence interval: Community,  $d = 1.11$ , 95%CI = 0.12 to 2.10, non-community  $d = 0.38$ , 95%CI = 0.05 to 0.71. Piquart et al. (2007) reported no correlation between treatment setting (inpatient versus other settings) and effect sizes ( $B = -0.19$ ,  $\beta = -0.14$ ,  $t = -1.29$ ).

#### **2.4.6.2.3. Gender (four analyses)**

Gorey and Cryns (1991) reported non-significance without supporting data. Engels and Vermey (1997) compared treatments where the proportion of women and men was fairly equal with treatments with a large proportion of women. They found a non-significant difference ( $Z = 1.69$ ,  $p = 0.6$ ,  $n = 17$ ) but note that the results are difficult to interpret as gender had to be coded at study, rather than intervention level and non-psychological interventions were included in the analysis. Cuijpers (1998) reported gender was not significantly correlated with outcome in a regression analysis across studies. Bohlmeijer et al. (2003) found no significance when comparing studies with more than 72% females with studies with less than 72% female, with 95% confidence interval overlapping: >72% female,  $d = 0.58$ , 95 % CI = 0.31 to 0.84, compared with, <72 % female,  $d = 0.75$ , 95% CI = 0.21 to 1.28.

#### **2.4.6.3. Factors for which the majority of analyses showed a moderating effect on treatment response**

##### **2.4.6.3.1. Outcome measures (8 analyses)**

Six studies found that clinician rated measures significantly increased effect sizes as compared with self-rated outcome measures. Engels and Vermey (1997) found the clinician-rated Hamilton Depression Rating Scale (HDRS) (Hamilton, 1967) significantly increased effect sizes as compared with all other outcome measures including self-rated outcomes such as the Becks Depression Inventory (BDI) (Beck, Brown, Steer, & Weissman, 1991) and the Geriatric Depression Scale (GDS) (Yesavage et al., 1983): HDRS  $d = 1.10$ ; BDI  $d = 0.57$ ; GDS  $d = 0.68$ . Paired t-tests showed this to be significant: HDRS vs. BDI:  $Z=3.30$ ,  $p=0.00$ ; HDRS vs GDS:  $Z=1.56$ ,  $p = 0.05$ . Piquart and

Sorensen (2001) also found that clinician rated measures significantly increased effect sizes as compared with self-rated outcomes across a wide range of interventions: CBT,  $ES(sr) = 0.64$ ,  $ES(cr) = 1.18$ ; Psychodynamic therapy,  $ES(sr) = 0.79$ ,  $ES(cr) = 1.68$ ; Reminiscence,  $ES(sr) = 0.44$ ,  $ES(cr) = 0.66$ . Pinguart et al. (2006) similarly found clinician rated depression to be associated with larger effect sizes for CBT:  $ES(sr) = -0.88$ ,  $ES(cr) = -1.22$ . Wilson et al. (2008) using the weighted mean difference (WMD) found that CBT was superior to active control as measured by the HDRS:  $WMD = -5.69$ ,  $95\%CI = -11.04$  to  $-0.35$ ,  $N = 86$ ) but not as measured by the GDS ( $WMD = 2.00$ ,  $95\%CI = -5.31$  to  $1.32$ ,  $N = 80$ ). Krishna et al. (2011) found that behavioural therapy was significantly more effective than waiting list control as measured by the HDRS ( $ES = -0.95$ ,  $95\%CI = -1.75$  to  $-0.14$ ) but not when measured by the BDI ( $ES = 0.19$ ,  $95\%CI = -0.82$  to  $0.45$ ) or the GDS ( $ES = -0.10$ ,  $95\%CI = -0.86$  to  $0.37$ ). Samad et al. (2011) found behavioural therapy for older people significantly more effective than wait-list (WL) control when measured by HDRS ( $WMD = -5.68$ ,  $95\%CI = -7.71$  to  $-3.66$ ,  $p < 0.001$ ,  $n = 117$ ) but not significantly different when measured by patient self-report measures GDS and BDI ( $SMD = -0.52$ ,  $95\%CI = -1.35$  to  $0.30$ ,  $p = 0.21$ ,  $n = 117$ ). Scogin and McElreath (1994) also report increased effect sizes associated with clinician rated outcome measures (mean  $ES(cl) = 1.15$ ,  $Z = 3.64$ ,  $p < 0.05$ , weighted mean  $ES(cl) = 1.33$ ) as compared with self-rated outcome measures (mean  $ES(sr) = 0.69$ ,  $Z = 3.31$ ,  $p < 0.05$ , weighted mean  $ES(sr) = 1.10$ ), however, they do not report the statistical significance of this finding.

Two studies found that outcome measures did not significantly predict effect sizes. Cuijpers et al. (2006) reported that effect sizes were comparable in studies using self-rating questionnaires and those where depression was defined according to diagnostic criteria, although the methodology for this analysis was not presented. Pinguart et al. (2007) reported overall effect sizes for self-rated and clinician rated depression overlapping at the 95% confidence interval:  $ES(sr) = 0.84$ ,  $95\%CI = 0.71$  to  $0.97$ ;  $ES(cr) = 0.93$ ,  $95\%CI = 0.74$  –  $1.11$ .

#### **2.4.6.3.2. Control used (six analyses)**

Four of the six analyses examining the impact of the type of control found that active or placebo controls were associated with reduced effect sizes when compared with no treatment or waiting list controls.

Gorey and Cryns (1991) reported group therapy versus no treatment, yielded an effect size of  $d = 0.68$ , compared with group therapy versus placebo  $d = 0.09$ . Unfortunately no details as to the process of classifying controls or describing placebo interventions were outlined in the methodology, thus making interpretation of this result difficult. Engels and Vermey (1997) reported that

comparisons with placebo yielded significantly lower effect sizes than no-treatment controls ( $Z = 2.08$ ,  $p = 0.02$ ,  $N = 16$ ) however they note that mean age varied significantly between these two conditions and non-psychological treatments were included in this analysis. Pincuart et al. (2007) also reported weaker improvement in depressive symptoms in interventions with active placebo control groups as compared with 'other' controls ( $\beta = -0.53$ ,  $t = -4.97$ ,  $p < 0.001$ ). Similarly, Krishna et al. (2011) found that behavioural therapy was effective for older people when compared with waiting list control (MD = 6.29,  $Z = 4.63$ ,  $p = 0.0001$ ) but not more effective when compared with active controls (MD = -0.2,  $Z = 0.18$ ,  $p = 0.86$ ).

Two studies found that type of control used did not significantly alter effect sizes. Cuijpers et al. (2006) found no significant difference in effect sizes between waiting list ( $d = 0.72$ ), care as usual ( $d = 0.75$ ) and 'other' controls ( $d = 0.64$ ). However, the study did not detail the nature of 'other' controls included, and did not undertake specific analysis between active placebo controls and non-active controls. Pincuart and Sorensen (2001) did not find a significant difference between changes in control groups using psychological placebo with changes in waiting list controls (self-rated depression:  $g = 0.12$ , 95% CI = 0.00 to 0.24, compared with  $g = 0.04$ , 95% CI = -0.03 to 0.11).

#### **2.4.6.3.3 Severity/presence of depression (nine analyses)**

##### **2.4.6.3.3.1. Increased severity of depression associated with larger effect sizes.**

Gorey and Cryns (1991) found that studies 'narratively defined' as including mild, moderate or severe depression showed significant variance in outcomes, with higher levels of depression associated with higher effect sizes (mild,  $d = 0.14$ ; moderate,  $d = 0.94$ ; severe,  $d = 1.37$ ). Bohlmeijer et al. (2003) similarly found participants with elevated depressive symptoms showed greater mean effect size ( $d = 1.23$ ) than those without elevated symptoms ( $d = 0.37$ ).

##### **2.4.6.3.3.2. Participants reaching diagnostic thresholds for depressive disorder compared with non-depressed participants.**

Pincuart and Sorensen (2001) found significantly larger effect sizes associated with depressed participants in comparison to non-depressed participants on both self-report and clinician rated measures. Depressed participants showed mean effect sizes of  $ES(cr) = 1.16$ ,  $ES(sr) = 0.70$ , compared with non-depressed participants:  $ES(cr) = 0.40$ ,  $ES(sr) = 0.31$ . Samad et al. (2011) found that only including studies with a formal diagnosis of depression in their analysis reduced the effectiveness of behaviour therapy as compared with cognitive therapy on self-rated measures (All studies, WMD =

0.23 versus only studies with formal diagnosis: WMD = 0.02) although this was not statistically significant and only included a small number of trials.

#### **2.4.6.3.3.3. Increased severity of depression associated with smaller effect sizes.**

Pinquart et al., (2007) found weaker improvements of depressive symptoms associated with studies including patients with diagnosis of major depression as opposed to other mood disorders: minor depression, mixed depressive symptoms and dysthymia ( $B = -0.26$ ,  $\beta = -0.22$ ,  $t = -2.21$ ,  $p < 0.05$ ).

#### **2.4.6.3.3.4. Depression severity showed no moderating effect.**

Four studies found that depression severity did not predict effect sizes. Scogin and McElreath (1994) reported similar mean effect sizes when comparing studies including severe depression (ES = 0.76) with those only including mild or sub-clinical depression (ES = 0.79). Engels and Vermey (1997) compared mild/moderate depression with severe depression and found depression severity to be a non-significant predictor of effect sizes in both controlled ( $Z = 0.44$ ,  $p = 0.33$ ) and all studies with artificial controls ( $Z = 0.83$ ,  $p = 0.20$ ). Cuijpers (1998) compared studies with a formal diagnosis of depression with studies without this requirement and found a non-significant difference between effect sizes:  $d = 1.25$  versus  $d = 0.9$ . The remaining meta-analyses did not undertake analysis of depression severity. Cuijpers et al. (2006) comparing studies including only patients diagnosed with major depressive disorder, (MDD) with studies including patients with a range of depressive symptoms did not find any significant difference (MDD,  $d = 0.84$ , 95 %CI, 0.56 to 1.11, versus 'Other'  $d = 0.67$ , 95% CI, 0.49 to 0.85).

#### **2.4.6.4. Factors which the majority of analyses found to be non- significant in predicting treatment outcome**

##### **2.4.6.4.1. Treatment type (14 analyses)**

Eight of the fourteen meta-analyses found that type of therapy did not predict treatment response as measured by effect sizes. Gorey and Cryns (1991) did not present methodology for subgroup analysis and it is therefore difficult to evaluate the validity of their findings. Scogin and McElreath (1994) independently compared cognitive and behavioural approaches with 'other approaches' (treatment orientations defined as: behavioural, cognitive, psychodynamic and eclectic). Cuijpers (1998) compared CBT with 'other therapies' and behaviour therapy with 'other therapies'. Bohlmeijer et al. (2003) compared reminiscence with life review. Cuijpers et al. (2006) compared CBT



to 'other treatments'. Wilson et al. (2008) compared cognitive with behavioural approaches and cognitive with psychodynamic approaches. Peng et al. (2009) compared CBT with reminiscence approaches. Samad et al. (2011) sub-divided the analysis according to self-rated and clinician rated outcome measures and compared behavioural therapy with cognitive therapy and behavioural therapy with brief psychodynamic therapy. None of the above comparisons revealed treatment type to be a significant predictor of outcome.

Four meta-analyses found that type of therapy was significant in predicting outcome. Engels and Vermey (1997) found that cognitive therapy (CT) and behaviour therapy (BT) were independently more effective than therapies which were defined as both cognitive and behavioural in their orientation (CBT). CT,  $d = 0.78$ ; BT,  $d = 0.85$ ; CBT,  $d = 0.12$ . Unfortunately the methodology for classifying treatment orientation was not adequately reported so it is difficult to evaluate the implications or validity of this finding. Engels and Vermey (1997) also found both cognitive and behavioural approaches to be more effective than 'other' therapies, but the latter category included poor quality studies examining non-psychological approaches making it difficult to draw any meaningful conclusions from this analysis.

Pinquart and Sorensen (2001) found that CBT and Psychodynamic approaches were associated with significantly higher effect sizes than reminiscence approaches: CBT,  $ES = 0.64$ , 95% CI = 0.5 to 0.78; Psychodynamic therapy,  $ES = 0.79$ , 95% CI = 0.37 to 1.21; Reminiscence,  $ES = 0.44$ , 95% CI = 0.30 to 0.58. Significant heterogeneity was noted in the findings for CBT but not psychodynamic therapy. Pinquart et al. (2007) found reminiscence, and cognitive behavioural approaches to show large effect sizes. Moderate effect sizes were found for psychodynamic therapy: Reminiscence,  $ES = 1.00$ , 95% CI = 0.73 to 1.27; Cognitive behavioural approaches,  $ES = 1.06$ , 95% CI = 0.87 to 1.26; Psychodynamic therapy,  $ES = 0.76$ , 95% CI = 0.31 to 1.21.

Two of the studies (Cuijpers, 1998; Samad et al., 2011) that did not find treatment type to be a significant predictor of outcome overall nonetheless found some evidence that treatment type was a significant variable. Cuijpers (1998) found CBT to be a predictor of increased effect sizes with multiple regression analysis whilst Samad et al. (2011) found that the effectiveness of behavioural therapy as compared to cognitive therapy was reduced (although this did not reach significance) when studies without a formal diagnosis of depression were excluded and outcome was measured via self-report measures.

#### 2.4.6.4.2. Modality (seven analyses)

Modality (group or individual) was found not to be related to treatment outcomes in five out of seven analyses (Bohlmeijer et al., 2003; Cuijpers, 1998; Cuijpers et al., 2006; Pincus et al., 2007; Scogin & McElreath, 1994). Two studies found therapy modality to be a predictor of treatment efficacy. Engels and Vermey (1997) found individual therapy to be more effective than group therapy (Individual  $d = 0.76$ , group  $d = 0.38$ ). However neither the relative quality of studies included in each category nor the proportion of psychological/non-psychological studies contained within each category were detailed, making it difficult to interpret this result. Pincus and Sorensen (2001), including only psychotherapeutic interventions, found individual interventions to be significantly more effective than group interventions with both self-rated and clinician-rated outcome measures: Individual,  $ES(sr) = 0.70$ ,  $ES(cr) = 1.56$ ; Group,  $ES(sr) = 0.44$ ,  $ES(cr) = 0.68$ .

#### 2.4.6.4.3. Age of participants (four analyses)

Three studies found that age was not significantly associated with effect sizes. Gorey and Cryns (1991) report non-significance without supporting data. Regression analysis undertaken by Cuijpers (1998) showed age to be a non-significant predictor of effect size across fourteen studies. Regression analysis by Pincus et al. (2007) also yielded non-significant results for age effects. Engels and Vermey (1997) employed a methodology which included artificial controls and note that the mean age of clients in the controlled studies was 70 years (range 65-81) and in the studies with artificial controls, 64 years (range 52-68). Including all studies they found a non-significant relationship between age and outcome ( $r = 0.03$ ,  $p = 0.41$ ,  $n = 28$ ). However, only including studies with real controls yielded a significant result with younger age associated with better outcome ( $r = 0.33$ ,  $p = 0.01$ ,  $n = 20$ ).

#### 2.4.6.4.4. Study quality (five analyses)

Three studies found that study quality was not associated with effect size. Scogin and McElreath (1994) found no correlation between study quality and effect size ( $r = 0.19$ ,  $n = 14$ ) in treatment versus no-treatment or placebo comparisons. Pincus and Sorensen (2001) divided studies into three quality categories and found no significant relationship between study quality and effect size (low  $ES = 0.35$ ; medium  $ES = 0.56$ ; high  $ES = 0.56$ ). Bohlmeijer et al. (2003) identified high quality studies and compared these with the remaining studies. They found that effect sizes overlapped significantly at the 95% confidence interval meaning study quality was not a reliable predictor of effect size: High quality:  $d = 0.92$  (95% CI = 0.28 to 1.56); other studies:  $d = 0.60$ , (95% CI = 0.33 to 0.88).

Two studies found a significant relationship between study quality and effect sizes. Gorey and Cryns (1991) reported that effect sizes attenuated by two thirds as study quality increased from the lowest to highest:  $F(3,18) = 9.90, p < 0.001$ . Pinquart et al., (2007) similarly found studies of higher quality were associated with lower effect sizes.  $B = -0.23, t = -2.25, p < 0.05$ .

#### 2.4.6.5. Factors only examined by one study

Gorey and Cryns, (1991) found smaller groups ( $<6, d=1.38$ ) to be more effective than larger groups (6-14,  $d = 0.81$ ) and found that studies with more than a quarter of participants living alone yielded a greater effect size than studies where less than a quarter lived alone ( $d = 1.42$ , compared with  $d = 0.84, p = 0.041$ ). However, methodology to control for potential confounding factors was not evidenced. Pinquart & Sorensen (2001) examined the impact of therapist variables on outcome. They divided interventions into those delivered by: graduate level therapists or paraprofessionals; therapists with advanced degrees; therapists with advanced degrees plus significant gerontological experience. The latter category was associated with significantly higher effect sizes ( $N = 108$ , mean  $ES = 0.81, 95\% CI = 0.56$  to  $1.06$ ) as compared with the other two categories. There was no statistical significance found between the effect sizes associated with both graduate level paraprofessionals ( $N = 161$ , mean  $ES = 0.28, 95\% CI = 0.07$  to  $0.28$ ) and those with advanced degrees ( $N = 316$ , means  $ES = 0.40, 95\% CI = 0.24$  to  $0.56$ ).

#### 2.4.7. Predictors of drop-out

Six studies included analysis of drop-out. Gorey and Cryns (1991) found that studies with drop-out ranges of 0-15 % produced a higher mean effect size ( $d = 1.26$ ) than studies with 16-50% drop-out ( $d = 0.31$ ). They infer that subject attrition was likely selective: those who may benefit most being also those more likely to drop out. Cuijpers (1998), transforming the binary drop-out data to undertake a regression analysis, found four significant variables which accounted for 94 % of the variance ( $F = 28.47, p = 0.0002$ ): group interventions ( $B = 0.36, SE = 0.06$ ); CBT interventions ( $B = 0.29, SE = 0.05$ ); treatments with a higher percentage of female participants ( $B = 0.02, SE = 0.003$ ); interventions offering more sessions ( $B = 0.04, SE = 0.01$ ). Variables that were not associated with increased drop-out included individual therapy, behavioural therapy bibliotherapy, pre-test BDI scores and age. Pinquart et al. (2007) also found higher dropout rates in group interventions ( $B = 0.49, \beta = 0.36, t = 2.45, p = 0.05$ ) and in longer interventions ( $B = 0.01, \beta = 0.25, t = 1.99, p = 0.05$ ). Mean age, type of control condition, study quality, depression severity, therapy setting and presence of co-morbidities were not correlated to drop-out.

Wilson et al. (2008) found that CBT was associated with higher drop-out when compared with control conditions (OR = 0.43). However this comparison showed significant heterogeneity ( $\chi^2 = 14.9$ ,  $p = 0.01$ ) and when compared with active controls CBT was not associated with higher drop-out (OR = 1.19). Comparing cognitive therapy with behavioural therapy and CBT with psychodynamic therapy, treatment approach was not shown to predict drop-out. Peng et al. (2009) pooled the results of drop-out across five studies examining treatment with anti-depressant medication with or without psychotherapy and found that adjunct psychotherapy did not predict drop-out (OR = 1.03,  $p = 0.92$ ). Samad et al. (2011) calculated the pooled drop-out ratio of cognitive therapy versus behaviour therapy (OR = 2.04, 95% CI = 0.87 - 4.78,  $p = 0.10$ ) and for behavioural therapy versus brief psychodynamic therapy (OR = 1.50, 95% CI = 0.32 - 6.96,  $p = 0.61$ ). Both results were non-significant.

#### **2.4.8. Relevant evidence from studies not included in the systematic review.**

A non-systematic meta-analytic review by Kiosses et al. (2011) examined predictors of treatment outcome and moderators of treatment effect in late-life major depressive disorder. They reviewed four studies. Depression severity did not moderate outcome in two studies (Alexopoulos et al., 2011; Arean et al., 2010), but showed significance in the other two: Thompson et al. (2001) found that patients with high baseline depression, compared with patients with low baseline depression, may have improved outcomes when treated with a combination of CBT and desipramine as opposed to desipramine alone. van Schaik et al. (2006) found that patients with a high baseline depression showed increased response to interpersonal therapy as compared with patients with low depression scores. Elsewhere, in a meta-analysis which did not limit its criteria to late life depression, Payne and Marcus (2008) found group psychotherapy to be less effective with older adults than with younger cohorts. A recent meta-regression analysis which did not limit its analysis to late-life depression, found no differential efficacy between CBT, problem solving therapy and inter-personal therapy for both younger and older adults with depression. Employing a multivariate analysis which controlled for participant characteristics, intervention and study design, no significant difference between psychotherapy efficacy for younger and older adults was found, although the authors note that heterogeneity was high in most analyses undertaken and warn that caution should therefore be exercised in interpretation of these results (Cuijpers et al., 2009).

Table 4: Summary of main findings and further analyses for included studies

	Main Finding	Further Analyses
<b>Scogin &amp; McElreath (1994)</b>	Psychosocial interventions more effective than no-treatment or placebo in decreasing depressive symptoms in older adults. Effect size: 0.78	Neither type of therapy, severity of depression, therapeutic modality, duration of therapy nor study quality found to be associated with effect sizes.
<b>Engels &amp; Vermey (1997)</b>	Overall effect sizes: Cognitive therapy $d = 0.78$ ; Behaviour therapy, $d = 0.85$ ; Cognitive and Behaviour Therapy, $d = 0.12$ ; Psychodynamic therapy, $d = 0.61$	Individual rather than group treatment and use of clinician rated outcome (HRSD) rather than self-rated (BDI/GDS) measures were significantly associated with greater effect sizes. Comparisons with placebo yielded significantly lower effect sizes than no-treatment or waiting list controls. No evidence of moderating effect for severity, age, gender, length of treatment. Those with multiple complaints were found to respond less well to treatment. Age: The mean age in controlled versus uncontrolled studies differed significantly. Including artificial controls in analysis yielded non-significant correlation.
<b>Cuijpers (1998)</b>	Psychological treatment for depressed elders in the community is effective: $d = 0.77$ .	Regression analysis showed CBT to be a predictor of increased effect size. Format, number of sessions, depression severity, gender and mean age were non-significant predictors. CBT not significantly more effective when compared with other approaches. Drop out significantly larger in: group interventions; CBT interventions; interventions with more sessions; conditions with a greater percentage of women.
<b>Pinquart &amp; Sorensen (2001)</b>	CBT and Psychodynamic approaches were associated with significantly higher effect sizes than reminiscence approaches: CBT, $ES = 0.64$ ; Psychodynamic therapy, $ES = 0.79$ ; Reminiscence, $ES = 0.44$ . However, significant heterogeneity was found in most analyses. The main effect for psychodynamic therapy (self-rated measures) was not subject to significant heterogeneity.	Individual interventions, Interventions with depressed, rather than non-depressed elders and use of clinician rated (HRSD) rather than self-rated (BDI/GDS) outcome measures were significantly associated with greater effect sizes. Therapists with advanced degree plus gerontological experience associated with significantly higher effect sizes as compared with those with just advanced degree / graduate / paraprofessionals. Type of control, study length, study quality and setting were not found to moderate effect sizes.
<b>Bohlmeijer et al., (2003)</b>	Overall effect size for Reminiscence/life review therapy $d = 0.84$ although test for heterogeneity indicated significant variance attributable to the systematic effects of covariates.	Larger effect in subjects with increased depressive symptoms as compared to other subjects. All other sub-group comparisons were non-significant and overlapped at 95% CI: Reminiscence versus life review; high versus low quality; group versus individual; community versus non-community; studies >72% women versus <72% women; published versus unpublished.
<b>Cuijpers et al., (2006)</b>	Psychological treatments have moderate to large effects on late-life depression. Overall effect $d = 0.72$ .	Equivalence of effect found between individual, group or bibliotherapy formats and between CBT and other types of psychological treatment. Severity of depression did not predict outcome. The effects were comparable between self-rated and clinician-rated depression outcomes. No impact of control group on effect size found.

<b>Pinquart et al., (2006)</b>	Psychotherapy and pharmacotherapy did not show strong difference in effect sizes: Both moderately large.	Greater clinician-rated depression improvement seen in mild to moderate depression, for psychotherapy group as compared with drug therapy. This was not found on self-rated measures. Effect size for CBT as rated on clinician-rated measures was greater than the effects of other forms of psychotherapy. Results for self-rated measures show similar trend but with significant heterogeneity.
<b>Pinquart et al., (2007)</b>	Self-rated depression $d = 0.84$ . Clinician-rated $d = 0.93$ . Both showed significant heterogeneity. CBT and reminiscence yielded large effect sizes. Psychodynamic therapy a medium effect size.	Weaker improvements of depressive symptoms were found in studies with active control group; physical co-morbidity; cognitively impaired patients; major depression (versus other mood disorders); studies of higher quality. Age, format, duration, outcome measure used and treatment setting did not show treatment effects. Higher dropout rates found in group interventions and in longer interventions.
<b>Wilson et al., (2008)</b>	CBT more effective than waiting list controls. (WMD -9.85, 95% CI -11.97 to -7.73) No significant difference between psychodynamic therapy and CBT.	Bibliotherapy more effective than waiting list controls. CBT superior to active control when using the HRSD, but equivalent when using the GDS. Treatment approach did not predict dropout.
<b>Peng et al., (2009)</b>	CBT, Reminiscence and 'General Psychotherapy' more effective than placebo/no intervention in decreasing depression scores.	CBT: SMD= -1.34. 95% CI, -1.89 to -0.79. Reminiscence: SMD= -0.64. 95% CI, -1.04 to -0.25. 'General Psychotherapy': SMD= -1.00. 95% CI, -1.40 to -0.59.
<b>Krishna et al., (2011)</b>	Group psychotherapy was an effective intervention for late-life depression compared with waiting list controls (very modest effect size). Group intervention versus active interventions did not reach statistical significance indicating no effect of the intervention versus all variations in the active control conditions.	Waiting list control had significantly fewer losses to follow up than intervention groups. HDRS outcome measure found significant difference favouring the group therapies compared to active controls but this was not found with BDI and GDS outcome measures.
<b>Samad et al., (2011)</b>	Behavioural therapy for older people significantly more effective than waiting list control when measured by clinician-rated measure (HRDS) WMD = -5.86 95%CI -1.35 to 0.30 but not significantly different when measured by patient self-report (BDI & GDS): WMD = -0.52 95%CI -7.71 to -3.66.	Excluding studies without a formal diagnosis of depression at baseline reduced the effectiveness of cognitive therapy compared to behavioural therapy (In self-reported depression).

## 2.5. Discussion

### 2.5.1. Moderating factors: patient characteristics

With regards to moderators of treatment associated with patient characteristics, this study confirmed results from previous reviews that have found no overall effect of gender or age on therapeutic outcome (Department of Health, 2001b), but did not add significantly to our understanding of how chronicity, relapse, and recovery may show variation across the age range (Alexopoulos et al., 1989). Apart from age and gender, few patient characteristics were routinely

coded by the included meta-analyses, reflecting the general paucity of such data historically reported in primary studies. Clinical guidelines frequently identify the importance of tailoring therapeutic interventions to fit with an individual's unique presentation, however, research into the impact of patient characteristics is not well-developed with much evidence from single studies without replication (Department of Health, 2001b).

Beutler et al. (1991) found that depressed patients' predisposing coping styles, significantly predicted differential response to differing treatment modalities. In a small trial comparing 63 patients with major depressive disorder, 'Externalising' patients improved more than 'internalising' patients in cognitive therapy, whereas the latter improved more in response to supportive, self-directed therapy. 'High defensive' patients showed greater improvement in supportive, self-directed therapy, whilst 'low defensive patients' improved more in cognitive therapy. Lower educational achievement has been found to increase the risk of discontinuation in self-directed therapy, whereas higher scores on a measure of 'learned resourcefulness' is a predictor of improved outcome in such approaches (McKendree Smith & Floyd, 2003). Whisman (1993) found that depressed patients with higher dysfunctional attitude scores showed poorer outcomes in cognitive therapy, whilst Piper et al. (1998) found that reduced capacity for interpersonal relating was found to be associated with poorer outcomes in psychodynamic therapy, as compared with supportive therapy.

Brand and Clingempeel (1992) reported that patients who had higher baseline positive social behaviours, combined with reduced physical co-morbidity and increased contact with family members, showed most benefit from group behavioural therapy for late-life depression. Allowing patient's to choose therapeutic modality was found to reduce drop-out, but had no impact on outcome (Rokke et al., 1999). Little evidence currently exists with regard to the management of older adults who would meet criteria for a diagnosis of personality disorder (Payman, 2011).

Meta-analytic investigation of which patient characteristics may function as moderators is limited by the data collected by primary studies. When factors such as ethnicity, socio-economic status and personality factors are not comprehensively detailed in primary studies, meta-analyses are limited in the claims that can be made with regard to the generalisability of intervention effects. Severity of presenting problem is the most frequent patient characteristic examined in moderator analyses. Results from this review yielded contradictory results as to the relationship between effect sizes and the severity of depression. The method of classification of depression severity within studies is a likely confounding factor in this analysis and may explain some of the inconsistency. It is not possible to establish for example, whether studies defined narratively as 'mild' depression in one study equate to a diagnosis of 'dysthymia' in another study. The analysis by Samad et al. (2011)



revealed diagnosis of depression to show interaction with treatment approach, with inclusion of studies with a criteria of major depression reducing the comparative efficacy of behavioural therapy as opposed to cognitive therapy. However, very few studies were included in the analysis and it did not reach statistical significance. The inconsistency of results regarding the impact of depression severity on outcome found by the current review is reflected in the wider literature.

Increased severity of depression has been found to be associated with slower response to treatment (Dew et al., 1997; Gildengers et al., 2005), poorer outcomes (Karp et al., 2005; Watt & Cappeliez, 2000; Thase et al., 1997) or has not shown any significant relationship with outcome, (Mintz et al., 1992; Lenze et al., 2001; Robinson et al., 1990). More recently, Driessen et al. (2010) undertaking a 'number needed to treat analysis' (Kraemer & Kupfer, 2006) found that, for less severely depressed patients compared with controls such as pill placebos, clinicians would need to treat eight patients, whilst for severe depression this number reduced to only three patients.

Poorer health has been found to be predictor of less successful outcome in younger adults but not in older adults (Harpole et al., 2005; Hughes et al., 1993). This finding was not supported by this current review, which found some limited evidence that co-morbidity may attenuate effectiveness of interventions for late-life depression. However these findings were based on only two post-hoc analyses and so little can be inferred with regard to the specific mechanism by which co-morbidities may moderate treatment efficacy and claims with regard to the generalizability of these findings must be tentative at best.

### **2.5.2. Moderating factors: treatment characteristics**

The absence of a relationship between treatment duration and treatment effect found by this review is perhaps surprising but is supported by previous findings (Molenaar et al., 2011; Robinson et al., 1990). This review of meta-analyses did not find consistent support for the finding that individual therapy is generally more effective than group approaches (Cuijpers, van Straten & Warmerdam, 2008). In addition, treatment setting was not found to have any measurable independent impact on treatment outcomes. Only one study examined the impact of therapist experience, finding specific gerontological expertise was associated with greater clinical improvement (Pinquart & Sorensen, 2001) a relationship also found by Cuijpers et al. (2008) undertaking a meta-regression analysis of characteristics associated with effective psychological treatments of depression, who found that studies with less experienced therapists yielded lower effect sizes. However in Pinquart and Sorensen's study (2001) it is not clear to what extent possible confounding factors such as therapeutic alliance or adherence to treatment protocol were controlled for, making it difficult to critically



evaluate claims made by other authors that variance within treatments due to therapist factors is greater than variance between treatments (Crits-Christoph, 1997; Wampold & Serlin, 2000).

### 2.5.3. Treatment approach

Results from this review of meta-analyses indicate that neither direct comparisons between treatment approaches nor meta-regression analyses yield consistent evidence for the superiority of any one therapeutic approach. The failure to find differential treatment effects might be due to factors associated with primary studies: they may be underpowered to detect the differential impact of treatment approach (Kazdin & Bass, 1989; Norcross, 1995) or fail to adequately control for important non-specific factors (Baskin et al., 2003). Alternatively it might be that the 'active ingredients' proposed by the various treatment approaches to be responsible for amelioration of depressive symptoms may not be the primary factors in effecting change. The proposed meditational effect of depressogenic cognitions posited by cognitive therapy for example, have been demonstrated in analyses of mediating factors (Knoop et al., 2012). However, such mediation has not consistently been found in the general adult population and the mediational effects of dysfunctional attitudes, as measured by the Dysfunctional Attitudes Scale (Beck et al., 1991) has found to be reduced in older adults (Whisman, 1993).

Reasons proposed to explain this age difference have included the impact of common factors and but also the specific need to address hopelessness in late life depression (Floyd & Scogin, 1998). Blazer (2003) in his comprehensive review of the literature for late-life depression proposes that the equivalence of therapeutic efficacy found for various treatment modalities, may in fact be the result of psychotherapeutic approaches sharing a central core mechanism: that of developing meta-cognitive awareness (Teasdale et al., 2002). This is the process by which patients 'step-back' from negative cognitions and begin to respond to them as mental events, rather than as the inherent aspects of the self.

Other possible explanation for the discrepancy between the differential treatment effect frequently reported in primary research, and the broad equivalence of effect often reported in meta-analytic studies include the impact of investigator allegiance. Robinson (1990), reviewing the literature across the age-range for treatments of depression, found that apparent differences in efficacy between treatment modalities disappeared once the moderating factor of investigator allegiance was included in the analysis. Although the adequacy of randomisation and concealment was explored in a minority of included meta-analyses, Investigator allegiance was not explicitly analysed as a potential moderating factor in any of the meta-analyses included in this review.

#### 2.5.4. Factors associated with study design

Two of the more robust findings of this study do not relate to variation in the clinical intervention or the participant but the increase in effect sizes associated with use of clinician rated outcome measures rather than self-rated measures and the use of waiting list control groups rather than active or placebo controls. The latter finding is consistent with results from a recent meta-regression analysis examining the characteristics of effective treatments for depression (Cuijpers et al., 2008) which found that effect sizes were reduced in studies using treatment as usual or placebo controls as opposed to waiting list controls. Although these findings do not directly shed light on what might work for whom, they do highlight the need to critically evaluate the reactivity of outcome measures and the adequacy of psychological controls when interpreting claims with regard to treatment efficacy of specific interventions.

Another important factor to consider is the significant heterogeneity seen in most of the meta-analyses seeking to differentiate between treatment approaches. This heterogeneity suggests that treatment effects are, to some significant extent, associated with uncoded and possibly confounding factors that remain poorly understood (Matt & Navarro, 1997).

#### 2.5.5. Drop-out

With regard to predictors of drop-out, two meta-analyses found that patients were more likely to drop out of group interventions rather than individual interventions but again it is not possible to discern the impact of possible confounding factors or determine specific factors associated with drop out (Davis & Hooke, 2006). Further replication of this finding would be required to facilitate conclusions with regard to its generalizability.

#### 2.5.6. Limitations of this study

The aim of this review was not to systematically review what is currently known with regards what works for whom in late life depression, but rather to explore to what extent meta-analyses have informed our understanding of this question. Systematic criteria were applied in order to limit the risk of reporting bias. However, the focus on meta-analytic studies meant potentially relevant studies were excluded. Examples include the non-meta-analytic review by Kiosses et al. (2011) examining predictors of treatment outcomes and moderators of treatment effect in late-life major depressive disorder. Similarly evidence from individual studies reporting predictors of outcomes and moderating factors were also excluded, as was evidence from the expanding literature

seeking to develop and adapt evidence-based psychotherapeutic interventions for older adults in the context of conceptual and theoretical constructs of ageing (Knight & Poon, 2008; Laidlaw, 2001; Laidlaw & McAlpine, 2008; Satre et al. 2006). This is a rich and expanding area which to date has been theoretically driven, with only limited useful data emerging from meta-analytic studies that could inform clinical and process issues within therapy.

The failure of this review to include an independent researcher to search, retrieve and screen studies introduces a significant risk of bias. Reporting bias may also have been introduced to the methodology of this study through the use of PRISMA checklist as a framework for evaluation of study quality and risk of bias. Although a proportion of studies were independently assessed according to the PRISMA guidelines quality rating was potentially a significant source of reporting bias due to the subjective nature of this process.

The significant relationship found between publication date, increased reporting quality and reduced analysis of predictors of outcome/moderating factors, indicates that reporting bias is likely an important factor in critically evaluating the data. Two potential hypotheses could explain such a finding. It may be that older studies, in the absence of quality reporting guidelines and with considerable pressure on word-limits, may have simply failed to systematically report sound methodology, focusing rather on reporting moderator analyses. Similarly, more recent meta-analyses, with an increased requirement to report methodological factors systematically, may simply have omitted examining moderating factors due to pressures of space.

Alternatively, if we hypothesize a true correlation between methodological rigour and reporting rigour, we might conclude that sub-analyses were less frequent in later studies due to the increased cognisance of the risks of type 1 and type 2 errors when undertaking sub-group analyses in relatively small heterogeneous data sets. As such, unplanned post-hoc analyses that were potentially undertaken in earlier studies may have been avoided in later, better quality studies.

How we interpret the relationship between date, reporting quality and number of moderator analyses influences the degree of scepticism we bring to evaluating the validity of moderator analysis in earlier studies. If we conclude the relationships are better explained as an artefact of reporting conventions rather than methodological improvements, then we may be at risk of overvaluing potentially unreliable data. Conversely if we believe that fewer moderator analyses were undertaken in more recent meta-analyses due to appropriate methodological constraints being observed, then we risk undervaluing earlier data that may indeed be robust. The present analysis rather than weight studies according to reporting quality, has therefore sought to highlight some of the difficulties of

such weighting by undertaking regression analyses to draw attention to the relationship between these interconnected factors.

Another source of potential bias, and a limitation to this study was the focus on depression ratings as the only measure of treatment effect. It could be argued that a narrow focus on symptom reduction prevents comparison of treatment efficacy across other potential treatment goals in therapy, such as quality of life, self-efficacy, personal insight or functional independence (Philips, 2009). It could also be argued that a narrow focus on symptom reduction places essentially heterogeneous depressive presentations along a reductive severity dimension, limiting the capacity for differential treatment responses to be identified (Parker, 2004).

Choosing an inclusion criterion of studies with a mean age of  $\geq 55$  yrs must also be considered a significant limitation of this study. This was undertaken because research in this area has tended to recruit 'young-old' samples. The alternative, more appropriate, cut-off at  $\geq 65$  yrs would have meant excluding the majority of studies undertaken into late-life depression. Indeed, none of the included meta-analyses set age inclusion criteria of  $\geq 65$  yrs. Referring back to Table 1, part 4, we can see that only three studies used a  $\geq 60$  yrs cut-off, with eight studies using an age of  $\geq 55$  yrs or lower. Indeed, three of the studies which met the criterion of mean age of participants of  $\geq 55$  yrs, actually set their own inclusion criteria at  $\geq 50$  yrs. Although this current study and previous analyses (Department of Health, 2001b) indicate that age may not be a significant predictor of therapeutic outcome, it is not clear whether there may be other consequences and limitations of using data from 'young-old' cohorts to understand late-life depression. This systematic problem in late-life depression research therefore means generalizing from current data to real-life clinical settings must be undertaken with great caution.

Finally, this review excluded meta-analyses which focused explicitly on treatments for depression in the context of significant medical co-morbidities. In light of the finding that the presence of co-morbidities showed a consistent impact on effect sizes (albeit with only two included analyses), the inclusion of studies that specifically addressed depression in the context of co-morbidities would have broadened the relevance and scope of the studies.

### **2.5.7. Implications of this review**

This review highlights a number of important factors for healthcare providers, users, and policy makers. Firstly, psychotherapeutic interventions for late-life depression consistently show moderate to high effect sizes thus efforts should be made to increase access to psychological therapies for older adults who, to date, have experienced inequitable access to psychotherapeutic interventions

as compared with younger adults (NHS Scotland, 2011; Age Concern, 2008; Bartels et al., 2002). Secondly, this study demonstrates that our current understanding of moderating factors in treatments for late-life depression is very limited. As such, this study points to the need for primary studies in this area which employs larger sample sizes, systematically reports a much wider range of patient and intervention factors and includes dismantling or additive designs that adequately attempt to quantify the impact of potential moderating factors (Ahn & Wampold, 2001). Thirdly, this study highlights a significant relationship between reporting quality of meta-analytic studies investigating psychotherapeutic interventions for late-life depression, and both date of publication and number of sub-analyses undertaken. It therefore highlights the need to critically evaluate claims with regard to differential treatment efficacy made by included studies in light of potentially higher risk of bias in older studies. Lastly, this review identified that psychotherapy effects appear to be attenuated when outcome studies employ self-rated rather than clinician-rated outcome measures and when active placebo controls are employed as opposed to comparisons with a no treatment groups. Implications for study design and critical appraisal of outcome studies, include the need to further investigate and refine psychological placebo conditions in order to more effectively discriminate differential efficacy with intervention groups, (Baskin et al., 2003; Serfaty et al., 2011) and the need for authors to critically appraise the sensitivity and suitability of outcome measures and their potential impact on estimations of effect size (Helmreich et al., 2011).

Developing an empirical understanding of the specific factors which make psychotherapy effective is far from straightforward. Ever since Smith & Glass (1977) undertook the first meta-analysis in this area and found negligible differences in the effects produced by different types of therapy, there has been a lively critical debate as to what exactly is working, and for whom, when psychotherapy is evidenced to be effective. Two broad schools of thought have emerged: those who claim that the evidence for differential efficacy of differing approaches is poor and that most psychotherapeutic approaches demonstrate broad equivalence in efficacy due to shared and powerful 'common factors' (Lambert, 2005; Luborsky, 2002; Messer, 2002; Wampold, 2005) and those which claim that the evidence for differential efficacy is well-established and linked to 'specific factors' that can be codified and systematically applied, often using treatment manuals (American Psychological Association, 1993; Chambless, 2002; DeRubeis & Crits-Christoph, 1998; Derubeis et al., 2005; Waltz et al., 1993; Wilson, 1996). The latter 'specific factor' paradigm is perhaps a better fit with the empirical methods and ontological assumptions of pharmaceutical research which seek to isolate the effect of discrete biochemical agents and consider variables such as 'dose response', 'active ingredients' and 'placebo effects'.

Such language is now commonplace in psychotherapy research and points to shared methodological assumptions with regards to the ability to control for moderating and mediating factors and ascribe changes in the dependent variable (usually reduction in symptoms) to changes in the independent variable (usually treatment type). However, adequately controlling for hidden confounding factors in psychotherapy research is far from straightforward (Dunn & Bentall, 2007). In practice, this means it has been easier to demonstrate a particular intervention works in a particular circumstance than it has been to demonstrate that a 'common factor' is the shared independent variable across varying approaches in differing circumstances. It could be argued that rejecting the null hypothesis that specific ingredients are not responsible for treatment effects (Wampold, 2005) requires modelling treatment effect heterogeneity in such a way that treatment outcome can reliably and independently be ascribed to discrete and measurable variables. It could be further argued that such rejection of the null hypothesis is an important first step in understanding not only 'what' is working in psychotherapy but also 'how' it might be working.

One perhaps unfortunate consequence of the methodological complexity that such an understanding seems to demand is the reductive focus on 'treatment type' and the emergence of what some have called a 'search for winners' (Stiles et al., 1986) with competing 'brand-name' therapies (Scogin et al., 2005) seeking to demonstrate relative superiority over each other. This emphasis on type of therapy has tended to de-emphasize the dilemma that establishing causality is far harder than demonstrating mediator status (Kraemer et al., 2002) and perhaps hindered rigorous research which seeks to experimentally define and manipulate both moderating and mediating factors in order to better understand potential common factors.

Meta-analytic investigation is not well suited to shedding light on the specific mechanisms of psychological change involved in treatment efficacy. Nonetheless to the extent that meta-analyses can reveal information about what might predict and moderate treatment response for specific disorders in specific patient groups they promise to facilitate the discrimination between moderators of treatment and mediators of treatment: i.e. separating those factors which might be the necessary conditions for a treatment to work from those factors which help elucidate how and why treatments work (Kraemer et al., 2002). Other approaches are better suited to examining questions with regard to mediating factors. Dismantling studies separate the existing components of effective therapies and seek to quantify to what extent each component is responsible for clinical change whilst experimental studies manipulate discrete independent variables thought to be active agents in therapeutic change and record any correlation with symptom change (Watkins, 2009). The use of dismantling studies and designs tailored to identify potential moderating and mediating factors (Kuyken et al., 2010; Labelle

et al., 2010; Lemmens et al., 2011; Warmerdam et al., 2010) are useful recent developments in psychotherapy research which may increase our ability to reject the null hypothesis that specific ingredients are not responsible for treatment effects.

The first systematic analysis of the efficacy of psychosocial interventions for late life depression concluded that it could be reported with some confidence that psycho-social interventions were effective for depression in late-life, but that the 'elusive active ingredients' and mechanisms of this efficacy were still poorly understood (Scogin & McElreath, 1994 p.73).

## 2.6. Conclusion

The current review has sought to assess to what extent meta-analyses have increased our understanding of what predicts or moderates treatment effects in late-life depression. In summary, it has failed to find consistent or robust data indicating a clear role for identifiable factors that might predict or moderate treatment efficacy, neither has it found a robust link between outcome variance and specific treatment approaches. With regards the question of how reliably evidence from controlled research can be generalized to clinical populations, (Barkham et al., 2008) the current review found that little information can be gleaned with regard to the specific needs of people of differing socio-economic status, those of differing ethnicity or cultural heritage, or the older old. This study has therefore highlighted, not only a need for more high quality studies to be undertaken with clinically representative older populations (Shadish et al., 2000), but also for future research to systematically explore both potential moderating and process factors within their experimental design .

- No consistent relationship between study quality and outcomes.
- No consistent relationship between depression severity or age and outcomes.
- Treatment setting, duration and gender of participants did not moderate outcomes.
- Patient-rated measures yielded smaller effect sizes than clinician-rated measures.
- Comparison with active controls yielded smaller effect sizes than waiting list controls.

### **2.7. Highlights**

(3-5 bullet points, maximum 85 characters, including spaces, per bullet point: See Appendix 1)



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**SECTION 3: Thesis Hypotheses**

### 3. Thesis Hypotheses

The preceding systematic review excluded meta-analyses examining psychotherapeutic treatments for older adults with specific underlying somatic diseases. Such studies were excluded to reduce the potential confounding impact of specific illnesses on the assessment of general treatment outcomes and moderating factors in psychotherapeutic treatments for late-life depression. However, late-life depression most frequently occurs in the context of chronic medical illness and cognitive impairment (Alexopoulos, 2005) and randomized controlled trials such as those included in the previous study, are not typically composed of clinically representative samples. It could therefore be argued that the findings of the previous analysis are limited with regard to drawing generalized conclusions about a clinical population where co-morbid illness is common.

Clearly, understanding the relationship between co-morbidity and depression is a key goal in effectively designing interventions for this age group. Cognitive behavioural therapy has been the most comprehensively evaluated intervention for late-life depression and has the strongest evidence base amongst psychological therapies (Bartels *et al.*, 2003; Gatz, 2007; Laidlaw, 2001; Scogin *et al.*, 2005). Numerous studies have been undertaken examining the efficacy of CBT to treat depressive symptoms in older adults with co-morbid physical illnesses. However, to date there has been no attempt to synthesize this data in a meta-analysis.

One of the challenges of undertaking such an analysis is the considerable heterogeneity in patient characteristics and intervention that such a synthesis would likely involve. However, one of the benefits of undertaking such a study would be its potential to inform our understanding of the effectiveness of CBT for depression in older adults across a diversity of physical illnesses, and as such, present data with regards the generalizability of CBT to real-life clinical settings.

The preceding review found some limited evidence to suggest that co-morbidity may moderate treatment efficacy in late-life depression. The meta-analysis in section 5 therefore aims to examine the effectiveness of CBT for late-life depression in people with a diversity of underlying physical illnesses. Results will be discussed in light of comparative evidence developed with AWA and critically evaluated with regards to validity issues associated with the meta-analytic methods undertaken.

**SECTION 4: Psychotherapeutic interventions for late-life depression: Evidence-based practice in context.**

## **4. Psychotherapeutic Interventions for Late-life Depression: Evidence Based Practice in Context.**

### **4.1. Introduction**

Evidence based practice (EBP) has been described as the integration of individual clinical expertise with the best external clinical evidence from systematic research (Sackett, 1996). The aim of EBP is to increase the application of clinical interventions that are known to be effective, and identify and end practices that are discovered to be ineffective. Empirical support for clinical interventions can be derived from a wide-range of sources. Well-designed, sufficiently powered randomized controlled trials which have been replicated by independent investigators represent the best primary evidence for the efficacy of any particular intervention. Systematic and meta-analytic reviews of such studies further extend our understanding of potential moderating factors and enhance our ability to draw conclusions about how such data can be generalized to broader clinical populations.

However, although such studies are vital in developing an empirical understanding of what might work for whom in clinical practice, in many areas, including late-life depression, there are considerable practical difficulties in implementing such rigorous methodologies with representative patient groups. Such limits have meant that controlled trials of interventions for late-life depression have disproportionately tended to recruit young-old, healthy, white participants, (Karel & Hinrichsen, 2000) limiting the degree to which the evidence generated by such studies can be reliably generalized to real life clinical settings.

Organisations such as the American Psychological Association (APA) National Institute for Health and Clinical Excellence (NICE) and the Scottish Intercollegiate Guidelines Network (SIGN) have been key drivers of the implementation of evidence-based practice. Clinical guidance produced by such bodies presents best-practice recommendations based on systematic reviews of the evidence base. However, the absence of evidence for a particular intervention is by no means evidence of its ineffectiveness, and inevitably the evidence-base for some psychological therapies has been more extensively studied than for others. Although the erroneous conflation of lack of evidence with ineffectiveness is explicitly acknowledged and warned against in such guidance, it has been argued that there has been a disproportionate emphasis on particular therapeutic modalities being endorsed by organisations such as NICE (New Savoy Partnership, 2011). Gaps in the evidence base not only occur due to the absence of eligible trials for specific approaches, but also frequently reflect a poorness of fit between the demands of empirical criteria and the actual clinical needs being met in health and social care settings. Margaret Gatz (2007) highlights the fact that in practice settings with older adults, combinations of pharmacotherapy and psychotherapy may be frequent, interventions

for emotional disorders may involve concurrent environmental adaptations and systemic interventions with care-givers, and individuals may present with a range of physical and cognitive co-morbidities.

Tailoring interventions to best meet the clinical needs of a diverse and heterogeneous client group such as depressed older adults, therefore involves not only drawing upon evidence generated from high quality clinical trials, but also integrating insights from gerontological theories of aging and understanding to what extent evidence generated for other groups, for example adults of working age (AWA), can be applied to this population. Synthesizing individual clinical expertise with the best external clinical evidence from systematic research therefore requires an understanding of some of the unique challenges faced by depressed older adults and specifically what might distinguish late-life depression from depression in other age groups. The next section will therefore examine important contextual factors which might differentiate the experience and presentation of depression in older adults, before reviewing the evidence-base for late-life depression and those approaches considered to have been demonstrated as efficacious treatments.

#### **4.2. Late-life Depression: A Complex Picture**

Laidlaw (2001) has warned against the perception that depression is a natural response to old-age. This 'fallacy of good reasons' (Unützer *et al.*, 1999) and the resulting risk of 'therapeutic nihilism' is perhaps beginning to be challenged (Teri *et al.*, 2004) but may be a factor in understanding why depressed older adults continue to receive poorer care than their younger counterparts (Bartels, 2002). Iliffe (2009) points out that seeing late-life depression as an understandable response to the challenges of aging risks confusing the natural human response of 'sadness' with clinically significant depressive symptoms. However, making sense of emotional suffering in late-life involves acknowledging the many challenges that may be encountered. Older adults face an increased likelihood of experiencing physical ill-health, dementia, chronic pain and neuropsychological changes. In addition role transitions, social isolation, and personal loss are often more salient. Contextual stressors, such as the increased likelihood of experiencing long-term residential or hospital care are also common. Sadavoy (2009) draws attention to the complex and reciprocal interplay between personality, physiology, neuroimmunological changes, environment and life events that need to be considered when assessing and treating late-life depression. He describes the five C's of working with older adults as complexity, chronicity, co-morbidity, continuity and context: highlighting the need for clinicians to be able to formulate a sensitive and detailed understanding of late-life depression in light of a broad range of relevant factors. Fiske *et al.* (2010) similarly describe a 'life span developmental diathesis stress model' and emphasize that, to adequately account for the reduced prevalence of major depression seen in

later years, there is a need to understand both risk and protective factors associated with advancing years.

#### 4.2.1. Prevalence and severity of depression in late-life

Depression is a heterogeneous and broad diagnosis and estimates of prevalence vary considerably depending on the severity of depression assessed and measures used. A number of depressive disorders are described in the Diagnostic and Statistical Manual for Mental Disorders: fourth edition (American Psychiatric Association, 2000) and the International Classification of Diseases 10<sup>th</sup> revision (World Health Organisation, 1992) ranging from major depressive disorders to mild depression and dysthymic disorders. Reviewing thirty-four studies, Beekman *et al.* (1999) presented estimates of depression in community dwelling older-adults ranging between 0.4 and 35 per cent. A prevalence of 1.8 per cent for major depression and 9.8 per cent for minor depression was found, whilst depressive symptoms deemed 'clinically significant' yielded an average prevalence of 13.5 per cent. 'Clinically significant' in this context remains somewhat ill-defined. Some researchers, finding that functional impairment increases linearly with symptom severity, identify clinically significant symptoms above a certain, sub-syndromal, threshold on depressive symptom checklists (Judd & Akiskal, 2002). Such approaches propose a dimensional rather than categorical quality to depressive conditions (Slade, 2007). Other taxometric investigations conversely support a categorical model of major depressive disorder (Ruscio *et al.*, 2007).

Such nosological questions as to whether depression is a homogenous (dimensional) disorder or heterogenous (categorical) disorder are relevant because community dwelling older adults consistently show reduced prevalence rates for major depression as compared with those found in adults of working age (Hasin *et al.*, 2005; Kessler *et al.*, 2003), whilst also showing increased rates of sub-threshold, clinically significant symptoms (Fiske *et al.*, 2010). It has been proposed that this pattern is may be artefact of the diagnostic criteria for major depressive disorders which emphasize dysphoria: a symptom less readily endorsed by older adults, whilst requiring clinicians to exclude symptoms that may be attributed to recent bereavement or medical condition, both factors more common in later years (Fiske *et al.* 2010) Conversely, depressive symptom checklists also often include symptoms associated with ill-health or bereavement, possibly increasing the risk of inflated sub-threshold depression scores (Blazer, 2003). Others have argued that reduced prevalence rates observed for major depression, rather than reflecting artefacts of diagnostic practices or differences in cohort attributions, actually reflect protective factors associated with ageing (Blazer, 2010).

Despite the ongoing nosological debate the impact of clinically significant depressive symptoms are not in doubt. Blazer (2003) reports approximately 15 per cent of community dwelling older adults to be experiencing clinically significant depressive symptoms, whilst a report of the surgeon general (U.S. Department of Health and Human Services, 1999) noted a higher prevalence and impact of 'sub-threshold' symptoms characterising this age group. Sub threshold symptoms are often chronic (Beekman *et al.*, 1999) and an estimated 8-10 per cent of older people with such symptoms go on to develop 'major depression' (Blanchard, 1996). For older adults, the burden of 'sub-threshold' symptoms can be as disabling as major depression, with comparable levels of functional impairment (U.S. Department of Health and Human Services, 1999), disability days and impact on self-rated health (Blazer, 2003; Hybels *et al.*, 2001). The increased recognition that depressive symptoms below the DSM-IV and ICD-10 threshold criteria can have considerable impact on functioning is now reflected in evidence-based treatment recommendations (National Institute for health and Clinical Excellence (NICE), 2009). Sub-threshold symptoms have been found to be correlated with a prior history of depression, neuroticism, poor physical health and disability (Blanchard, 1996) and occur more commonly in long-terms care settings than in community settings (Meeks *et al.*, 2011).

Beekman *et al.* (1999) found higher prevalence rates of depression for women and those older adults experiencing adverse socio-economic circumstances. Prevalence rates of 16 per cent were found in a London inner-city sample where around two-thirds of the sample group were female (Livingston *et al.*, 1990) Medical burden, low social support and disability have also been identified as important risk factors (Meeks *et al.*, 2011) with depression estimated to be at least twice as frequent among patients in hospital or nursing homes (Baldwin & Wild 2004) with a recent report by Age Concern (2008) estimating that two in five care home residents experience depression. Gellis *et al.* (2007) found a prevalence rate of 13.7% for major depression and 27.5% for clinically significant depressive symptoms in community dwelling older adults receiving care at home. Osborn *et al.* (2002) found a prevalence rate of 13.1 per cent for community residents aged 75 and over, whilst Stek *et al.* (2004) found even higher prevalence rates (15.4 per cent) in the oldest-old living in the community. For this older group depression was correlated with cognitive and functional impairment. However, although depressive symptoms appear more commonly in the oldest old, when prevalence rates are adjusted to account for the higher proportion of women, physical disability, lower socio-economic status, and cognitive impairment, no significant correlation between increased age and depression is found (Blazer *et al.*, 1991)



In summary, whilst rates of major depressive disorder seem to be less prevalent in older adults, the burden and prevalence of mild and sub-threshold symptoms may be disproportionately higher in this age group. Despite the high prevalence of such clinically significant depressive symptoms in older adults, the detection rate of depression in primary care has been found to be very low, (Crawford *et al.*, 1998) with depression remaining worryingly under-diagnosed and under-treated (Age Concern, 2008; Bruce *et al.*, 2002; Lebowitz *et al.*, 1997; Royal College of Psychiatrists, 2011; Wilson *et al.*, 2008).

#### 4.2.2. Cohort specific risk factors

Factors which increase the risk of developing depression for AWA continue into later life, including being female, socially isolated, unmarried, poor, and having a previous history of depression (Bisschop *et al.*, 2004; Meeks *et al.*, 2011; Unützer *et al.*, 1999) Further risk factors disproportionately faced by older adults include loss and bereavement, sleep disturbance, and disability (Cole & Dendukuri 2003) with the risk of depression particularly elevated amongst elder carers of others with serious medical or conditions (Russo *et al.*, 1995). The death of a spouse increases the risk of developing major depression and impacts particularly on older adults who have reduced social support or live alone (Knight & Poon, 2008). The increased likelihood of significant physical comorbidity and neurobiological change are also significant factors.

#### 4.2.3. Cohort beliefs, expectations and attributions

Today's older adults seek psychological support less readily than their younger counterparts but this may be changing as attitudinal differences have been observed across older adult cohorts, with younger members expressing more positive attitudes to mental health services than their elders (Currin *et al.*, 1998; Segal *et al.*, 2005). It is relatively rare for older adults with depression to be offered psychological interventions (Baldwin & Wild 2004) but one large scale RCT found that, when treatment options are offered, at least half of older people experiencing depression expressed a preference for psychological treatment over drugs (Unützer *et al.*, 2002): a preference confirmed by a number of previous studies (Areán & Cook, 2002; Landreville *et al.* 2001; Rokke & Scogin 1995). Landreville *et al.* (2001) found that severity of depressive symptoms affected the acceptability of differing treatment modalities: Cognitive therapy and cognitive bibliotherapy were rated as more acceptable than anti-depressant medication for patients with mild to moderate depression, whilst cognitive therapy was rated as more acceptable than both anti-depressant medication and cognitive bibliotherapy for severe depression. Similarly, Hanson and Scogin (2008) found that older adults expressed a preference for combination of psychotherapy and anti-depressant medication rather than medication alone for treatment of late-life depression.

In a study comparing community based cognitive behavioural interventions with younger and older adults, older adults demonstrated a relative preference for non-pharmacological interventions and had significantly better attendance and drop-out rates. Efficacy of intervention was comparable across the age range including those over seventy-five (Walker & Clarke, 2001). The authors note that the relative increased engagement of older adults in this study may have been due to the availability of home assessment. Practical and physical barriers to engagement have been proposed as contributing factors to the inequitable service provision and relative underutilization of mental health services in late life (Yang & Jackson 1998).

Compared with adults of working age, older adults are more likely to underestimate and downplay depressive symptoms or ascribe them to physical complaints (Blazer, 2003; Robb *et al.*, 2003). They are also less likely to report sadness as the predominant feature during a depressive episode (Lebowitz *et al.*, 1997). Among the older-old (80+ years) subjective well-being is less correlated with physical and functional health than for those between 60-80 years of age. In fact, for the older-old, subjective well-being and mental health are more closely correlated than for their younger counterparts (Pinquart, 2001).

#### 4.2.4. Risk of suicide

Suicidal ideation is closely associated with severity of depressive symptoms in older adults (Alexopoulos *et al.*, 1999) with major depression a significant predictor of suicide in this age group (Waern *et al.* 2002). Older adults are almost twice as likely to commit suicide than AWA (Alexopoulos, 2005) and this elevated risk is largely due to increased rates amongst elder white males. (Kung *et al.*, 2008) Although suicidal ideation decreases with age, older people are more likely to act on such thoughts with fatal consequences (Beeston, 2006; Conwell *et al.*, 1998; Conwell *et al.*, 2002). Depressive syndromes are found in 80 per cent of those over 74 years who commit suicide (Conwell *et al.*, 1996) and the risk of suicide is shown to be increased for those experiencing minor depression or dysthymic disorder (Conwell *et al.*, 2002). Hawton and Harriss (2006) following up 700 older people admitted to hospital following a suicide attempt, found physical health problems to be the most frequent life problem associated with the admission (46.1%). Social isolation, relationship problems and bereavement and loss are other common life problems associated with suicide attempts in older adults (Harwood *et al.*, 2006; Hawton & Harriss, 2006) with disruption of inter-personal relationships associated with risk of suicide independently of depression severity (Alexopoulos, 2005). Hirsch and Duberstein (2009) seeking to understand the relationship between suicidality, depression and physical health problems in older adults, found that amongst almost 2,000 primary care patients,

positive mental health was a significant protective factor in reducing suicidality for older adults experiencing physical health problems.

#### 4.2.5. Co-morbidity and neurological changes

Perhaps the most important factor to understand in assessing and treating late-life depression is its relationship with physical illness and disability. Functional impairment and medical co-morbidity increases the risk of depression across a wide range of disorders including diabetes (Blazer, 2002) chronic obstructive pulmonary disease (Yohannes *et al.*, 1998) heart conditions (Ariyo *et al.* 2000) Depression predicts both cardiac morbidity and mortality (Carney 2003) and has been found to be associated with reduced bone mineral density, and increased risk of osteoporosis (Robbins *et al.*, 2001). A number of possible physiological mechanisms for the association between depression and chronic ill-health have been explored. Depression often involves reduced appetite which can lead to reduced resilience and frailty (Blazer, 2003). A 'vicious cycle' has been proposed in which chronic pain may cause reduced deep stage sleep, worsening depressive symptoms, which then in turn further impact on sleep patterns thus reducing energy and motivation and to undertake protective activities such as moderate exercise (Unützer *et al.*, 1999). Increased platelet activation has been observed in depressed patients, indicating a possible mechanism for increased risk of ischaemic damage (Whyte *et al.* 2001) whilst impaired immune response has also been observed in elders experiencing chronic mild depressive symptoms (Blazer, 2003).

Patients experiencing their first episode of depression in later life are likely to have a more chronic course than those of the same age with a recurrent presentation, and such late onset depression has been associated with neurobiological changes, specifically white matter hyperintensities and ventriculomegaly (Lebowitz *et al.* 1997) Possible neurobiological links have been proposed between stroke and depression (Baldwin & Wild 2004) and around a fifth of Alzheimer patients are estimated to experience major depression (Blazer, 2003). Depressive symptoms thought to be linked to vascular changes in the brain, so called 'vascular depression,' (Alexopolous *et al.*, 1997) have been linked with specific deficits in executive functioning (Steffens 2004) and other structural brain changes including enlargement of lateral ventricles; cortical atrophy; increased likelihood of basal ganglia lesions and reduced putamen and caudate (Unützer *et al.*, 1999). Co-morbid cognitive dysfunction increases risk of mortality in depressed older adults (Kane *et al.*, 2010). Some authors have proposed that medical co-morbidities such as Parkinson disease, thyroid pathologies and diabetes, amongst others, can function to mask depression (Colasanti *et al.*, 2010) with depressive symptoms erroneously ascribed to medical complaints. It is suggested that this 'masked depression' can be particularly important when

considering that older people who may meet the criteria for a depressive condition are themselves more likely to report somatic complaints rather than mood disturbance (Christensen *et al.*, 1999).

#### 4.2.6. Cohort specific protective factors

Wisdom has long been a quality traditionally associated with advancing years, and more recently a subject for empirical study (Baltes & Staudinger, 2000). Despite the high incidence of physical illness, social isolation, and personal losses often encountered in late-life, lower rates of major depression are consistently found in older adults as compared with AWA. Theories of wisdom and emotional development across the lifespan have been one means to understand this apparent paradox (Blazer, 2010).

Baltes & Staudinger (2000) have defined and operationalised qualities which might be associated with wisdom: recognising and managing uncertainty; placing concerns and worries within a lifetime temporal perspective; acknowledging and accept the relativity of values; drawing upon a rich store of procedural and factual knowledge to solve problems. Such qualities might be understood to moderate the impact of difficult life circumstances and represent important protective factors that may develop with advancing years. (Windle & Woods 2004) found that a sense of environmental mastery mediated the impact of deteriorating physical health and housing concerns on overall life satisfaction whilst Baltes and Baltes (1990) have proposed that 'successful ageing' may involve the selection of appropriate goals, the optimization of current skills to minimise losses and the adoption of alternative strategies to compensate for changing abilities (SOC model). Socioemotional selectivity theory (Carstensen *et al.*, 2000) proposes that limits on perceived available time leads to the prioritizing of emotional goals: with increasing years, comes greater awareness of the finitude of life and an increasing motivation (and hopefully capacity) to engage with goals or activities which are emotionally rewarding in the present rather than activities which defer satisfaction in the service of long-term goals or responsibilities. One consequence of the development of such skills may be improvements in emotional regulation. Older adults have been found to be less reactive than younger adults to distressing event, particularly inter-personal stressors (Neupert *et al.*, 2007) and have been found to experience less affective reactivity than younger adults when faced with cognitively challenging tasks (Chow *et al.*, 2007).

In addition to demonstrating improved emotional regulation when dealing with the usual stressors of day-to-day life, Blazer (2010) draws attention to the fact that many of the more significant challenges of late-life are to some extent anticipated. As such older adults may be better prepared, both emotionally and practically, for personal losses and impaired functioning and thus demonstrate

greater resilience and acceptance. For individuals who are able to foster such positive attitudes towards late life, the potential challenges and losses may engender reduced cognitive dissonance and reduced subjective dissatisfaction with physical decline (Ron, 2007).

#### 4.2.7. Summary

Considering whether depression in late life is somehow distinct, it is perhaps important to acknowledge that whilst the qualitative experience of depression as an older adult may indeed vary from that of a younger adult, such differences should not be considered to be consequence of any normative developmental or aging processes but due to the fact that depressed elders face different challenges than their younger counterparts: They are more likely to experience medical co-morbidities, neurobiological changes, sleep disturbance, functional impairment, disability and loss, all of which increase the risk of developing depression. As we have seen, for those living in the community, depressive disorders are no more frequent in late life than in midlife (Blazer, 2003), however the impact of sub-threshold depressive symptoms may in fact be greater in this age group, and the risk of suicide much increased. For individuals experiencing long-term residential or hospital care the risk of depressive symptoms is much greater. As previously noted, research studies on late-life depression have tended to focus on relatively young, white, well educated, healthy cohorts (Karel & Hinrichsen, 2000) and there is therefore a need for studies which more specifically address how age-associated factors impact on late-life depression in 'real-life' community and clinical settings (Bartels *et al.*, 2003).

#### 4.3. Late-life Depression: The Current Evidence Base

As reported in the preceding systematic review, meta-analytic studies have found a range of psychotherapeutic interventions to be more effective than treatment as usual in treating late-life depression. Results from the review also show that, where analyses are undertaken comparing the relative efficacy of differing treatment approaches, they tend not to find evidence for the superiority of any particular approach. Possible reasons for this were touched upon in the preceding review and reflect the fact that the preponderance of outcome variance in trial data is not usually accounted for by the differing treatments themselves (Scogin *et al.* 2005). However, relative equivalence of efficacy in meta-analytic studies does not necessarily mean relative equivalence in the quality of evidence that generated this data. One way to assess the quality of empirical support for a particular intervention and so critically evaluate the outcome of such meta-analytic findings has been to establish criteria by which to code the empirical rigour of primary studies. Such criteria have been developed by the American Psychological Association in America (Chambless & Hollon 1998) and bodies such as NICE and SIGN in the UK. Interventions are assessed as 'evidence-based' depending on whether studies

demonstrating their efficacy reach certain quality thresholds and are adequately replicated (Yon & Scogin 2007). The first attempt to undertake an assessment of the evidence for late-life depression was undertaken by Gatz *et al.* (1998) using the American Psychological Association's criteria (Chambless & Hollon, 1998) for assessing efficacy of psychosocial interventions. They concluded behavioural, cognitive, brief psychodynamic therapy and life review/reminiscence were 'probably efficacious.' To reach the threshold for 'well established,' an intervention was required to out-perform a good quality psychological placebo or control treatment or demonstrate equivalence to an already well established intervention. A subsequent review by Areán and Cook (2002) found Cognitive Behavioural Therapy (CBT), Problem Solving Therapy (PST) and the combined effect of IPT with medication (CAMP-IPT) to be efficacious in treating ambulatory older adults with major depression, noting that brief psychodynamic therapy (BPT) required only one additional independent trial to be considered an efficacious treatment according to the criteria developed by Chambless and Hollon (1998). An update of this review (Mackin & Areán, 2005) concluded that BPT in addition to CBT, Reminiscence Therapy (RT), and the CAMP-IPT had achieved evidence-based status.

In the context of criticism that the American Psychological Association's original system was insufficiently codified or transparent, the guidance was refined and simplified from a two-tier model ('probably efficacious' and 'well-established') to a single-level endorsement of 'beneficial'. Scogin *et al.* (2005) applying these updated criteria identified six psychological treatments they considered showed sufficient evidence of efficacy to be classed as evidence-based treatments for late-life depression: behaviour therapy, cognitive behaviour therapy, cognitive bibliotherapy, problem solving therapy, brief psychodynamic therapy and reminiscence therapy. Subsequently, an independent interdisciplinary expert panel convening in April 2006 to develop recommendations for community-based treatment of late-life depression (Frederick *et al.* 2007) found that there was only sufficient evidence to recommend depression care management and individual CBT.

The development of evidence-based lists which create dichotomous groups of 'unsupported' or 'supported' treatments has been criticised on both pragmatic and theoretical grounds (Beutler, 1998; Westen & Bradley 2005). Westen *et al.* (2004) argue that data from meta-analytic studies consistently points to the need for a more nuanced assessment of treatment efficacy, and argue there is a poorness of fit between the demands of RCT methodology and most psychotherapeutic interventions (with the notable exception of exposure-based treatments for specific anxiety symptoms). Beutler (1998) points out that, in part, the endorsement of named, manualised therapies, emerged in response to changes in health care funding in the United States and was therefore not entirely driven by empirical or clinical demands. Scogin *et al.* (2005) point out that criticism of such practices has been somewhat less

vociferous in the area of gerontology and proposes that this has been due to the opportunity that such lists have provided to substantiate the efficacy of interventions with older adults in the context of a historical and inaccurate consensus which viewed psychological treatments with this group to be less effective than those provided to AWA.

Such lists run the risk of misrepresenting the breadth of available evidence, and reifying certain approaches that do not accurately address the complexity of real-life clinical circumstances, (Westen & Bradley 2005) perhaps consolidating a gap between efficacy (as established in controlled trials) and the effectiveness of treatments in real-life clinic settings (Unützer *et al.*, 1999). However, the American Psychological Association's guidelines for psychological practice with older adults (Teri *et al.*, 2004) are cognisant of such limitations and identify that no single treatment is preferable for depressed older-adults and that developing individualised treatment approaches requires consideration of a wide range of biological, psychological and social factors. Guidelines issued by NICE (2009) further note that such factors are not well captured by current diagnostic criteria and can have a significant impact on the course of depression and response to treatment.

Neither NICE nor SIGN have developed specific clinical recommendations for late-life depression, however clinical recommendations outlining a 'Matched/Stepped -Care' approach to treating late life-depression have recently been published as part of the MATRIX: A Guide to delivering evidence-based Psychological Therapies in Scotland (Scottish Government, 2011). These guidelines follow the hierarchical format of endorsing specific treatments according to the quality of evidence available, but authors note that such recommendations must be considered in the context of the conceptual and methodological constraints discussed above (K. Laidlaw, personal communication 2<sup>nd</sup> May 2012).

Having acknowledged some of the limits of developing such lists of evidence-based treatments, the next section will provide a brief outline of those treatments currently considered beneficial, and further identify other treatments for which there is a developing evidence base.

### **4.3.1. Evidence-based psychotherapeutic approaches for late life depression**

#### **4.3.1.1. Cognitive behavioural psychotherapies**

The term 'Cognitive Behavioural Therapy' can be used to describe a range of interventions derived from both behavioural and cognitive psychological models of human behaviour and development. Most frequently, both behavioural and cognitive components are combined in CBT interventions. However, both behaviour therapy and cognitive therapy are also practised as distinct disciplines. Behaviour therapy for depression draws upon theories of classical and operant conditioning and focuses on the relationship between subjective mood and pleasant and unpleasant events experienced



by the individual. It emphasizes the role of social learning and reinforcement in the maintenance of depressive symptoms and aims to reduce negative affect by developing skills in identifying, planning and increasing pleasurable activities. Cognitive therapy, on the other hand, draws upon extensive research into the mediating role of cognitions in the development and maintenance of emotional disorders. It is an active, time-limited and directive problem solving approach which involves identifying and monitoring distorted negative thinking, and applying techniques to challenge and moderate these thoughts. In both cognitive and behavioural approaches, individuals are expected to undertake structured tasks between sessions to consolidate and practice skills. In practice, CBT is often used to describe therapeutic interventions that are predicated on scientific principles and which pragmatically combine both behavioural and cognitive techniques.

#### **4.3.1.2. Cognitive behavioural therapy**

Reviews of the evidence base for late-life depression have consistently found cognitive behavioural approaches to have the most developed empirical base amongst psychological therapies for late-life depression (Bartels *et al.*, 2003; Gatz, 2007; Laidlaw, 2001; Scogin *et al.*, 2005). Scogin *et al.*, (2005) identified seven studies demonstrating the efficacy of CBT for late-life (Campbell, 1992; Floyd *et al.*, 2004; Gallagher & Thompson, 1982; Gallagher-Thompson & Steffen, 1994; May *et al.*, 2006; Rokke *et al.*, 1999; Thompson *et al.*, 1987). Since this review, an RCT undertaken by Laidlaw *et al.* (2008) found both treatment as usual and CBT to be effective treatments for mild to moderate late-life depression and Serfaty *et al.* (2009) undertaking the largest RCT of individual CBT for late-life depression in primary care to date, found CBT to an effective treatment as compared with a well-designed talking control and usual GP care. However a recent pilot randomised controlled trial failed to report effective reduction of depressive symptoms using a brief group cognitive behaviour therapy intervention with community dwelling older-adults (Wilkinson *et al.* 2009).

#### **4.3.1.3. Behavioural therapy**

Scogin *et al.*, (2005) identified five studies with a total of 111 participants, showing that behaviour therapy was superior control conditions and as effective as CBT or brief psychodynamic psychotherapy in treating late-life depression (Floyd *et al.*, 2004; Gallagher & Thompson, 1982; Lichtenberg *et al.*, 1996; Teri *et al.* 1997; Thompson *et al.*, 1987). A recent meta-analysis by Samad *et al.* (2011) (reviewed in the preceding systematic review) found behavioural therapy to be superior to wait list controls and as effective as cognitive therapy and brief psychodynamic therapy.



#### 4.3.1.4. Problem solving therapy

Problem solving therapy (PST) is a brief cognitive-behavioural approach typically between 4-6 sessions (Mynors-Wallis 2001) which seeks to improve patients' ability to understand the link between current symptoms and everyday problems and then learn skills to solve these in a structured way (Hawton & Kirk, 1989). PST proposes that deficits in problem-solving skills, specifically in interpersonal contexts, enhance the risk for developing depressive conditions. Arean *et al.* (1993), undertaking a randomised controlled trial, found problem solving therapy to be more effective than both reminiscence therapy and waiting list control for late life depression. It has also been shown to be more effective than supportive therapy in treating depression with older adults with executive dysfunction (Alexopoulos *et al.*, 2011) and an effective component of collaborative care in the treatment of depression with sustained long-term benefits when compared with treatment as usual (Hunkeler *et al.*, 2006). However, one study comparing the efficacy of paroxetine, PST and placebo pill over a course of six treatment sessions failed to find any benefit of PST over placebo (Williams *et al.*, 2000).

#### 4.3.1.5. Cognitive bibliotherapy

Cognitive Bibliotherapy (CB) is a self-directed treatment based on cognitive principles which involves the patient reading standardized treatment material and undertaking exercises focused on modifying maladaptive cognitive processes. In a review Scogin *et al.*, (2005) identified four eligible studies (Floyd *et al.*, 2004; Landreville & Bissonnette, 1997; Scogin *et al.*, 1987; Scogin *et al.*, 1989) with a total of 48 participants. All four studies found CB to be more effective than waiting list control for patients in the mildly depressed range. However, Landreville and Bissonnette (1997) found that post-treatment scores for the CB group remained in the mildly-depressed range and showed only slight improvement compared with untreated patients. In a two-year follow up to their 2004 study (Floyd *et al.*, 2004) comparing the efficacy of individual cognitive therapy and bibliotherapy, Floyd *et al.* (2006) found gains were maintained and equivalent for both forms of therapy, but found that relapse rates amongst the bibliotherapy group were significantly higher (5/11) as compared with the individual therapy groups (1/12). In conclusion, the efficacy of CB for moderate or severe depression has yet to be demonstrated (Frazer *et al.*, 2005) and bibliotherapy may be less effective than individual therapy in preventing relapse of depressive symptoms in older adults.

#### 4.3.1.6. Brief psychodynamic therapy

Brief psychodynamic therapy is a time limited approach, typically lasting three to four months (U.S. Department of Health and Human Services, 1999). Current problems are explored in the context of

prior developmental experiences, and current relationships. It draws upon psychoanalytic theory, examining unconscious motives, needs and defences and how these present within the dynamics of the client/therapist relationship. (Evans & Garner, 2004) Two studies have shown psychodynamic therapy to be as effective as other evidence based treatments such as CBT for treatment for late-life depression (Gallagher-Thompson & Steffen, 1994; Thompson *et al.*, 1987) with results maintained at follow up after two years (Gallagher-Thompson *et al.*, 1990). The efficacy of brief dynamic therapy compared with antidepressant medication has not been explored with older adults (Areán & Cook, 2002).

#### 4.3.1.7. Reminiscence therapy

Reminiscence therapy involves guided reflection upon both positive and negative life experiences with the aim of promoting cognitive and emotional engagement with self-narratives as a means to overcoming feelings of despair and low mood (Arean *et al.*, 1993). It is an intervention developed specifically for older adults and a number of studies support its application as an evidence-based treatment (Arean *et al.*, 1993; Serrano *et al.*, 2004; Watt & Cappeliez, 2000; Yen-Chun Lin *et al.*, 2003) In a meta-analysis Bohlmeijer *et al.*, (2003) (reviewed in section 1) found a large overall effect size for Reminiscence/life review but found significant heterogeneity: indicating significant variance attributable to the systematic effects of covariates. Reviewing the literature, Arean and Cook (2002) noted that controlled trials identified were underpowered, lacked formal diagnostic testing and often had small sample sizes but a recent RCT with 125 participants reported that group reminiscence therapy resulted in a significant reduction of depressive symptoms in community dwelling elders (Zhou *et al.*, 2012).

#### 4.3.1.8. Interpersonal therapy

Interpersonal therapy was developed as a time-limited treatment for depression in AWA and focuses on role disputes, role transitions, interpersonal deficits and grief. It has been proposed that it may be particularly suited to older adults given the higher likelihood of role changes, losses and social isolation in this group (Miller, 2008) and adaptations to the approach have been made to work with individuals with cognitive impairment (Miller & Reynolds, 2007). A number of reviews have identified Interpersonal therapy (IPT) as a probably effective treatment for late-life depression (Frazer *et al.*, 2005; Karel & Hinrichsen, 2000; Lebowitz *et al.*, 1997) However, although the evidence-base for IPT as a treatment for depression in AWA is considered well-established (SIGN, 2010; Scottish Government, 2011) there is a lack of studies demonstrating its efficacy for treatment of depression in older adults. Much of the IPT literature has examined its efficacy as a maintenance treatment in combination with medication or pill placebo, and as such it has been difficult to isolate

IPT's stand-alone efficacy or ascertain its effectiveness as an initial treatment for index episodes of depression. (Reynolds *et al.* 2010; Reynolds *et al.* 2006; Reynolds *et al.* 1999) The current evidence therefore indicates that IPT is effective as an adjunct to anti-depressant medication in reducing recurrence of depressive symptoms in late life, of but that further large RCTs are required for it to be considered an evidence-based stand alone treatment for depressed older adults.

#### 4.3.1.9. Other approaches

Other approaches, for which the data are not yet well developed, but which have shown some empirical support for treatment of late-life depression, include: Relational/Insight therapy (Gallagher & Thompson, 1982); Longer-term CBT and psychodynamic therapy (Steuer *et al.*, 1984); Personal Construct Therapy (Viney *et al.*, 1989); Behavioral Bibliotherapy (Scogin *et al.*, 1989); Family Therapy (Benbow *et al.*, 1990); Coping Skills Group Therapy (Dhooper *et al.*, 1993); Interpersonal Counselling (Mossey *et al.*, 1996); Goal-focused Therapy (Klausner *et al.*, 1998); Psycho-educational Groups (Schimmel-Spreeuw *et al.*, 2000) Dialectical behaviour therapy (Lynch *et al.*, 2007; Lynch, *et al.*, 2003)

In addition, long-term outcome results from large scale collaborative care approaches (Alexopoulos *et al.*, 2009; Hunkeler *et al.*, 2006; Unützer, *et al.*, 2002) have indicated the efficacy of enhanced care management tailored to individual needs which combine psychotherapeutic and behavioural interventions with anti-depressant medication. Such large scale, multi-site studies address some of the limitations of more typical RCT designs: they typically involve more representative samples and embed interventions within existing primary care services. As such, they potentially narrow the potential gap between experimental efficacy and real-life effectiveness (Unützer, *et al.*, 1999).

However, it is of course impossible to isolate the specific impact of the psychotherapeutic intervention in such approaches, and as such they have not been included in the current review.

#### 4.4. Summary

As reviewed in the preceding systematic review, meta-analyses have consistently found psychological treatments have moderate to large effects on late-life depression (Cuijpers, 1998; Cuijpers *et al.*, 2006; Engels & Vermey, 1997; Krishna *et al.*, 2011; Pinguart & Sorensen, 2001; Pinguart *et al.*, 2007; Samad *et al.*, 2011; Scogin & McElreath, 1994). Effect sizes are equivalent to those found for pharmacotherapy (Pinguart *et al.*, 2006) and studies with AWA (Cuijpers *et al.*, 2009) with individual therapy found to be more effective than group approaches (Cuijpers *et al.*, 2008). Cognitive behavioural approaches have been the most systematically evaluated and empirically validated approaches. Cognitive Bibliotherapy has shown efficacy for mild depressive symptoms, with

problem-solving therapy, behaviour therapy and cognitive behavioural therapy showing efficacy for more severe depressive presentations. IPT has demonstrated efficacy as an adjunct to anti-depressant medication and promising results with AWA indicate it is likely to be an efficacious treatment for older-adults, however further stand-alone RCTs are needed to confirm this. Studies of reminiscence therapy and life review approaches also show promising results but sufficiently powered, better quality trials are required to consolidate initial indications of its efficacy for late-life depression. BPT has been found to be as effective as CBT in a small number of RCTs, but more studies are required to establish a robust evidence-base for this approach.

There are limits of the generalizability of the data: further studies need to be undertaken in long-term care settings (Powers, 2008) with older adults with cognitive impairment (Areán & Cook, 2002) and the oldest-old (Blazer, 2003). In addition the evidence-base with regards to treatments of late-life depression in the presence of significant co-morbidities is not well developed. RCT's examining late life depression frequently use exclusion criteria based on 'clinical co-morbidity': when the presence of one disorder alters the normal course of the other (Stover *et al.*, 2003; McCusker *et al.*, 2005). However, the vast majority of older patients in primary care settings experience co-morbid illness with rates of depression increasing with higher co-morbidity (Charlson & Peterson 2002; NICE 2009). Frequently the degree and nature of co-morbidity in experimental samples is not well reported, meaning it is difficult to ascertain either the moderating impact of co-morbidities, or the relative efficacy of differing treatment approaches.

In light of these issues, the following meta-analysis will examine the efficacy of the best evidenced approach to depression in this age group, CBT, as it has been applied with older adults experiencing co-morbid physical illnesses.

**SECTION 5: Meta-Analysis (Journal Article)**

**A Meta-Analysis of Cognitive Behavioural Therapy for Treatment  
of Late-Life Depression in the Context of Physical Illness**

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**Abstract**

**Objective:** Examine the efficacy of cognitive behavioural therapy (CBT) for late-life depression in older adults with co-morbid physical illness.

**Method:** Systematic literature search and meta-analysis of randomised controlled trials (RCT) evaluating CBT for depression in older adults with co-morbid physical illness.

**Results:** Nine papers met inclusion criteria. CBT was superior to waiting list and treatment as usual control conditions, showing a statistically significant pooled standardised mean difference (SMD) of 0.63 (95%CI, 0.29 to 0.97,  $p = 0.0003$ ). This was largely maintained at follow up (SMD 0.5, 95% CI, 0.08 to 0.92). Sensitivity analysis showed individual CBT yielded a large, statistically significant summary effect size of 0.80 (95% CI, 0.45 to 1.16), but that group CBT did not show statistical superiority over controls. Clinician-rated measures of depression yielded larger effect sizes, with a SMD of 1.57 (95%CI, 0.56 to 2.59,  $p = 0.002$ ) as compared with patient-rated measures: 1.03 (95% CI, 0.75 to 1.31,  $p = 0.0001$ ).

**Conclusions:** CBT is effective in reducing depressive symptoms for depressed older adults with an underlying physical illness when compared with waiting list controls and treatment as usual.

Word count: 183/ maximum 200

Declaration of interest: None.

Key words: (maximum 6)

CBT, META-ANALYSIS, OLDER ADULTS, DEPRESSION, CO-MORBIDITY

### 5.1. Introduction

Depression in late-life frequently occurs in the context of co-morbid physical illness (Alexopoulos et al. 2002) and is associated with significantly reduced quality of life (Doraiswamy et al. 2002) poorer medical prognosis (Pennix et al., 2000) increased mortality (Kane et al. 2010) and significant increases in economic costs (Katon et al., 2003). Reported prevalence rates for major depression in medically ill older adults vary between 5% and 45% with rates for sub-syndromal or minor depression showing even greater variability (McCusker et al. 2005). Despite such variability, it is clear that physical co-morbidity greatly increases the likelihood of an individual becoming depressed (Alexopoulos, 2005; Charlson & Peterson, 2002; Fiske, Wetherell, & Gatz, 2010) with higher rates found in patients with cardiovascular disease (Carney, 2003), diabetes, (Lustman et al. 2000) stroke (Strober & Arnett 2009), Parkinson's disease (Reijnders et al. 2008) and Alzheimer disease (Park et al. 2007).

Psychological and pharmacological treatments have been found to be equally efficacious in treating late-life depression (Pinquart, Duberstein, & Lyness, 2006), but due to concerns with regards drug interactions, trials of anti-depressants have often excluded individuals with co-morbid physical illnesses (Stover et al. 2003). Psychological treatments may therefore be more suitable in this population and are certainly found to be frequently preferred by patients (Areán & Cook, 2002; Rokke & Scogin, 1995, Unützer et al., 2002). A number of previous meta-analytic studies have examined psychotherapeutic interventions for depression in individuals with physical illnesses (Sheard & Maguire 1999; Astin et al. 2002; Beltman et al. 2010; Dusseldorp et al. 1999; Himelhoch et al. 2007; Lustman et al. 2000; Linden et al. 2007; Meyer & Mark 1995; Tatrow & Montgomery 2006). Such studies have either limited their analysis to specific disorders, included a wide range of psychotherapeutic approaches or failed to distinguish between adults of working age (AWA) and older adults. Cognitive behavioural therapy (CBT) has the most developed empirical base amongst psychological therapies for late-life depression (Bartels et al. 2003; Gatz, 2007; Laidlaw, 2001; Scogin et al. 2005) and there is a growing body of evidence examining its application specifically in the context of medical co-morbidity. However, no meta-analyses have been undertaken examining the efficacy of CBT for this group. We conducted a meta-analysis examining the effectiveness of CBT for depression in older adults with co-morbid physical illness. We hypothesized that individual CBT would demonstrate similar efficacy to group approaches, (Cuijpers et al. 2008a) clinician-rated measures would yield greater effect sizes than patient-rated measures (Cuijpers, Li, Hofmann, & Andersson, 2010) and that depression severity would not moderate treatment efficacy (Driessen et al. 2010)



## 5.2. Methods

### 5.2.1. Identification of suitable studies

Electronic databases were searched until April 2012 using EBSCO host: CINAHL Plus; MEDLINE; PsycINFO; Psychology and Behavioral Sciences Collection; Biomedical Reference Collection. A boolean/phrase search mode limited to English language using text keywords with truncation and wild cards was used, structured as three concepts: Disorder (depress\* OR dysthymi\* OR mood); Intervention (psychotherap\* OR cognitive therapy OR behavi\* therapy OR CBT OR Problem Solving OR Stress Management); Design (Randomi?ed Controlled Trial OR RCT OR Controlled Trial). Terms relating to physical illness or age were not included to prevent exclusion of possibly relevant studies. Titles were screened to identify relevant studies. Somatic illnesses were identified. The Cochrane Library was searched, adding the dimension of specific underlying physical illnesses: (cancer OR COPD OR diabetes OR heart OR dementia OR Alzheimer\* OR coronary OR Parkinson\* OR arthritis OR HIV OR chronic health OR physical \*morbidity OR multiple sclerosis OR irritable bowel OR physical illness OR epilepsy). Reference lists of existing systematic reviews and of identified studies were hand searched, and authors of included studies were contacted. (Appendix 10 gives details of correspondence with authors). To minimise publication bias, a search of grey literature was undertaken (via [www.opengrey.eu/](http://www.opengrey.eu/)). To identify relevant ongoing clinical trials The World Health Organisation (WHO) International Clinical trials registry platform search portal was searched. Abstracts and full texts of included studies were screened for eligibility by one author (DH) in consultation with a second author (KL) (Full search strategy is outlined in Appendix 11).

### 5.2.2. Inclusion criteria

Studies meeting the following criteria were included<sup>1</sup>:

1. Use of a randomised controlled research design.
2. Mean age  $\geq 55$  years.
3. Inclusion of participants with an underlying physical illness.
4. Inclusion of a treatment arm with CBT, defined as a protocol- based clinician delivered intervention including clear well-described cognitive and behavioural components: problem-solving therapy, cognitive-behavioural stress management and mindful-based CBT interventions meeting these criteria were therefore eligible.
5. Treatment protocol described components explicitly focused on amelioration of depressive symptoms.

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<sup>1</sup> Guidelines devised by the Cochrane Collaboration were used to develop inclusion criteria (O'Connor, Green & Higgins, 2011)

6. Valid outcome measures used: self-report (e.g. Beck Depression Inventory (BDI); Beck et al. 1961), clinician-rated (e.g. Hamilton Depression Rating Scale (HDRS); Hamilton, 1967) or structured diagnostic interviews according to DSM-IV-TR criteria (American Psychiatric Association, 2000).

Studies were excluded where effect of CBT intervention could not be isolated from other treatment components such as anti-depressant medication or collaborative care management. Studies examining conditions without a definitive somatic origin were excluded (e.g. fibromyalgia, ME, pain management, or executive dysfunction in the absence of a diagnosed underlying illness). Studies employing guided self-help based on CBT principles were excluded.

### 5.2.3. Outcome measures

The primary outcome measure was change in depression symptoms assessed by clinician-rated (e.g. HDRS) or self-rated (e.g. BDI) measures using continuous data (mean and standard deviation). Validated self-report measures tend to yield smaller estimations of effect sizes in studies of late-life depression (Cuijpers, et al., 2010) and so a conservative approach was taken, with self-rated measures given precedence in studies where both were reported. Dropout rates from treatment were recorded as dichotomous data as a proxy for treatment acceptability.

### 5.2.4. Assessment of risk of bias

Risk of bias was assessed for each eligible study by two independent raters (DH & KL) using the Cochrane Collaboration risk of bias tool (Higgins & Green, 2008). Study Quality was assessed using the Cochrane Collaboration for Depression and Anxiety group Quality Rating Scale (QRS) (Moncrieff & Churchill, 2001) by two independent raters (DH & AL). The degree of the agreement between the authors was expressed as a percentage and a Kappa statistic. These tools were used to assess whether studies adequately concealed and randomized allocation to treatment, whether there was appropriate blinding of assessors, to identify risks of associated with incomplete data and selective reporting and assess the fidelity of interventions relative to described treatment protocols. Potential threats to study validity and risks of bias are discussed narratively.

### 5.2.5. Data extraction

For each eligible study, one author (DH) extracted information regarding methods, participants, intervention and outcomes. Data was checked by a second author (KL) and authors were contacted via e-mail to retrieve any missing data.

### 5.2.6. Data synthesis

To allow analysis of continuous measures of depressive symptoms across different scales, the standardized mean difference (SMD) between the intervention and control or comparison group was calculated for both clinician-rated and patient-rated scales where available. Mean scores and standard deviation on validated depression measures were used. The SMD expresses the size of the treatment effect for each trial relative to variability observed, enabling different scales to be pooled into one outcome measure. As Freedland et al. (2009) reported standard error scores with covariate-adjusted least-squares means, Cochrane Collaboration Review Manager Software (RevMan version 5.1, 2011) was used to transform standard error data into standard deviation data. Cohen's *d* (Cohen, 1987) was calculated by subtracting the average post-test score of the control group from the average post-test score of the experimental group and dividing the result by the pooled standard deviations of both groups. This was transformed into Hedge's *g* to adjust for possible bias resulting from small samples (Borenstein et al., 2009). Data were interpreted according to the convention: small (0.2) medium (0.5) and large (0.8) (Cohen 1992).

As heterogeneity was anticipated among the studies a random-effects meta-analysis was undertaken using the Cochrane Collaboration Review Manager Software (RevMan version 5.1, 2011). This involves the mean effect size of each study being weighted by the inverse of its variance to generate an overall weighted mean. Analyses were undertaken with intent-to-treat data where possible. Further details of methods are outlined in section 6.

### 5.2.7. Exploration of heterogeneity

The  $I^2$  statistic, which expresses the percentage of variability in an effect size that can be ascribed to study heterogeneity rather than to chance (Higgins & Thompson 2002), was used to explore statistical heterogeneity. Low heterogeneity is indicated by  $I^2$  values of 25%, moderate, 50% and high, 75% (Higgins et al. 2003). Potential sources of heterogeneity identified a priori were modality of intervention (group or individual) and depression severity at baseline. Sensitivity analyses were undertaken to assess the impact of sources of heterogeneity on overall treatment effects (Higgins & Thompson 2002). Depression severity at baseline was determined according to widely accepted thresholds on patient-rated measures: GDS-15 (Almeida & Almeida, 1999) BDI (Beck et al., 1961) and BDI-II (Beck et al., 1996). Due to anticipated variation in treatment delivery (group vs individual, number and length of sessions) data were pooled using a random effects model with 95% confidence intervals (Sutton et al., 1998). The likelihood of participants dropping out of the intervention group as compared with the comparison group was presented with odds ratios (OR). Publication bias was subjectively assessed by plotting the standardized mean difference (SMD) of

each study against standard error (SE) and reporting any observed asymmetry. Fail-safe N was calculated using methods outlined by Rosenberg (2005). Further details of methods are outlined in section 6.

### 5.3. Results

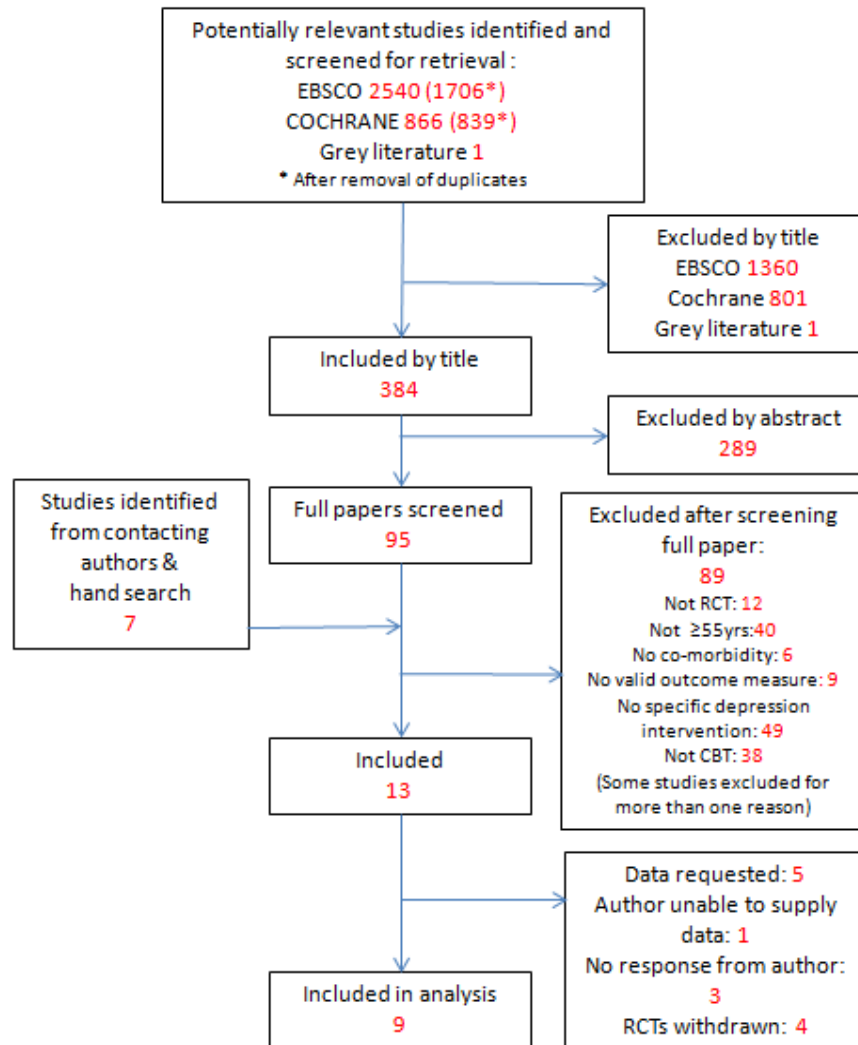
#### 5.3.1. Results of systematic search

A systematic search of electronic databases identified 2546 studies (Figure 1). Of these, 2162 were excluded by screening titles and 289 by screening abstracts. A total of 102 full papers were retrieved and assessed for eligibility. (Reasons for exclusion included as Appendix 12). Thirteen randomised controlled trials (RCT) were identified which met the inclusion criteria. Of these, 5 did not include sufficient data to allow meta-analytic synthesis. Attempts were made to contact the relevant authors. Two replied and one of these was able to supply the necessary data. Three could not be contacted.

#### 5.3.2. Study characteristics

The main characteristics of the nine included RCT's which collectively included 1104 participants are detailed in tables 1 to 3. Table 1 shows that the majority of studies undertook intent-to-treat analyses, used similar outcome measures and included patients of a similar age (mean: 63.6, SD 6.5) with moderate levels of depressive symptoms. There was considerable variation in the percentage of women in included studies and in the mode of delivery: Five studies examined individual, and four studies group interventions. Table 2 shows that the intensity of intervention also varied from four, hour-long sessions over three weeks (Dao et al. 2011) to 20 two-hour sessions over the course of a year (Koertge et al., 2008). Eight studies compared CBT to treatment as usual (TAU) (Dao et al., 2011; Dobkin et al., 2011; Freedland et al., 2009; Gellis et al., 2008; Hynninen et al., 2010; Koertge et al., 2008; Lincoln & Flannaghan, 2003) whilst Foley et al. (2010) used a wait-list control. Dobkin et al. (2011) and Hynninen et al. (2010) supplemented TAU with minimal telephone contact to monitor depressive symptoms. Lincoln & Flannaghan (2003) also employed an attention placebo comprised of ten hour-long visits from a clinician focused on discussions of day-to-day occurrences and the physical effects of stroke. Two studies compared CBT with another intervention. Freedland et al. (2009) included a stress management condition which involved weekly hour-long individual sessions focused on relaxation techniques such as controlled breathing and progressive muscular relaxation. Kunik et al. (2008) compared CBT with a COPD education intervention comprising eight hour-long sessions focused on disease management and education. Table 3 details the outcomes for each study.

Figure 1. Flow chart illustrating literature search process



Three studies were not included in analysis of follow up data because time from end of treatment to follow up was insufficient (Dao et al., 2011; Dobkin et al., 2011) or because follow up data was only reported for intervention group and not control (Foley et al., 2010).

### 5.3.3. Risk of bias in included studies

There was good agreement between the independent authors regarding study quality (88%, Kappa 0.75, 95% CI, 0.61 to 0.88) and the included studies achieved reasonable QRS scores of between 29 and 39 out of a maximum score of 46 with a mean score of 33.4 (SD 4.10) Agreement regarding risk of bias yielded a fair agreement (56%, Kappa 0.26, 95% CI, 0.04 to 0.47). Final ratings were agreed through discussion. (Authors' conclusions regarding risk of bias and study quality are detailed in Appendices 13 and 14).

Overall risk of bias can be seen in Figure 2. Adequate randomization was reported by seven studies, with risk of allocation bias unclear in Hynninen et al. (2010) due to insufficient information on procedure and in Koertge et al. (2008) due to the potential impact of pre-screening for participants' ability to attend twenty sessions. Five studies did not report sufficient detail of allocation concealment (Dao et al., 2011; Freedland et al., 2009; Hynninen et al., 2010; Koertge et al., 2008; Kunik et al., 2008). Due to the nature of the studies participant/clinician blinding was not possible, however, only two studies provided sufficient information to evidence that blinding of outcome assessors was undertaken in a way that would ensure detection bias was minimised (Dobkin et al., 2011; Foley et al., 2010).

Gellis et al. (2008) failed to employ intent to treat analysis in the presence of significant attrition indicating a high risk of attrition bias. Kunik et al. (2008), although using ITT methods, reported extremely high attrition without clear reporting as to reasons between groups and was considered, with five further studies to show unclear risk of attrition bias (Freedland et al., 2009; Hynninen et al., 2010; Koertge et al., 2008; Lincoln & Flannaghan, 2003; Foley et al., 2010). Dao et al. (2010) and Dobkin et al. (2011) showed a low risk for attrition bias, reporting low, non-skewed, attrition rates with clear reasons for drop-out. Pre-published trial protocols were not found for any of the included studies, meaning it was not possible to assess the risk of reporting bias.

Six studies were considered to show a high risk of bias due to other factors and these are recorded as 'Other Bias' in Figure 2. Hynninen et al. (2010) and Kunik et al. (2008) recruited using advertisements, with the resultant risk of non-representative samples. Failure to record, control or analyse potential impact for anti-depressant use was a significant risk of bias in five studies (Foley et al., 2010; Gellis et al., 2008; Hynninen et al., 2010; Koertge et al., 2008; Kunik et al., 2008). Inadequate test for treatment fidelity was evident in four studies (Gellis et al., 2008; Hynninen et al., 2010; Koertge et al., 2008; Lincoln & Flannaghan, 2003) and in the study by Lincoln and Flannaghan (2003) this was in the context of limited clinician training. Full details of all sources of bias identified, including 'other bias,' are listed in Appendix 13.

Table 1: Characteristics of Included Studies: Methods and Participants

Methods									
Authors (Date)	Blinding of outcome assessor	Intent to treat	Test of treatment fidelity		Control / †Comparison				
Lincoln & Flannaghan (2003)	Y. (Integrity not tested)	N*	Not described		TAU / †Attention placebo				
Gellis et al., (2008)	Y. (Integrity not tested)	N	Not described		TAU				
Koertge et al., (2008)	N. (Self-rated measures)	Y	Not described		TAU				
Kunik et al., (2008).	Y. (Integrity not tested)	Y	Y		†COPD education				
Freedland et al., (2009)	Y. (Integrity not tested)	Y	Y		TAU / †Stress management				
Foley et al., (2010).	Y. (Integrity tested)	Y	Not described		WL				
Hynninen et al., (2010)	N. (Self-rated measures)	Y	SPV but fidelity not confirmed.		TAU ( + minimal clinical monitoring )				
Dao et al., (2011)	N. (Self-rated measures)	N*	Y		TAU				
Dobkin et al., (2011)	Y. (Integrity tested)	Y	Y		TAU ( + minimal clinical monitoring )				
Participants									
Authors (Date)	Total	Female (%)	N			Age		Co-morbidity	Mean Depression at baseline
			CBT	Cont	Comp	Mean	SD / range†		
Lincoln & Flannaghan (2003)	123	49	39	41	43	UC: 65.0 AP: 66.1 CBT: 67.1	15.1 13.2 12.7	Stroke	'Moderate' (BDI)
Gellis et al., (2008)	69	88	36	33	-	77.4	2.3 / 65-99	Home-bound, medically ill.	'Severe' (HDRS)
Koertge et al., (2008)	247	100	119	128	-	62.1	8.9 / 35-75	Coronary Heart Disease	'Mild' (BDI)
Kunik et al., (2008).	238	4	118	-	120	66.3	10.2	COPD	'Moderate' (BDI-II)
Freedland et al., (2009)	81	50	41	40	42	CBT: 62 SSM 59 UC: 61	11 10 9	Coronary Heart Disease	'Moderate' (BDI) 'Severe' (HDRS)
Foley et al., (2010).	115	76	55	60	-	MCBT: 54.8 WL: 55.5	9.1 11.9 / 24-78	Cancer	Moderate (HDRS)
Hynninen et al., (2010)	51	51	25	26	-	CBT: 59.3 CG: 62.6	7.6 / 41-74 9.9 / 41-78	COPD	'Mild' (BDI-II)
Dao et al., (2011)	100	22	50	50	-	UC: 64.2 MADES:62.8	11.9 11.8	Coronary Heart Disease	'Moderate' (BDI-II)
Dobkin et al., (2011)	80	40	41	39	-	64.6	10.5	Parkinson Disease	'Moderate' (BDI) 'Severe' (HDRS)

KEY: \* Although no intent to treat analysis undertaken these studies had >95 completer data. † Only for those studies in which it was reported. AP = Attention Placebo. BDI = Beck's depression Inventory. CBT = Cognitive Behavioural Therapy. CG = Control Group. COPD = Chronic Obstructive Pulmonary Disease. HDRS = Hamilton Depression Rating Scale. MADES = Managing Anxiety and Depression using Education and Skills. MCBT = Mindfulness based CBT. SPV = supervision. SSM = Supportive Stress Management. TAU = treatment as usual. UC = Usual Care. WL = Waiting list.

Table 2: Characteristics of Included Studies: Intervention

Intervention					
Authors (Date)	Description	Setting Format	Session no. Duration Frequency	Attrition at end of treatment (%)	Follow-up (months after end of treatment)
Lincoln & Flannaghan (2003)	Manualised CBT: education, activity scheduling, graded task assignment, identification and modification of depressogenic thoughts. Delivered by single nurse therapist.	Home Individual	10 60min Over 3 months	4.0	3
Gellis et al., (2008)	PST. Involved education re: signs and symptoms of depression, developing specific problems solving and coping strategies. Using diaries and increasing daily pleasurable activities. Delivered by one PhD-level clinical social worker.	Home Individual	6 60 min Weekly	10.1	4.5
Koertge et al., (2008)	CBT stress management. Involved strategies to identify and moderate maladaptive cognitive, affective and behavioural patterns: assertive communication, strategic problem-solving skills, thought challenging and relaxation practice. Delivered by two nurse therapists.	Out patient Group (4-6)	20 120 min 10 weekly, then 10 monthly	15.4	17 (Mean; 1-2 years)
Kunik et al., (2008).	CBT for both anxiety and depression including psycho-education on anxiety and depression, behavioural activation, problem solving techniques, cognitive techniques, sleep management and planning for maintenance of gains. Delivered by psychology interns and post- doctoral fellows.	Out patient Group ( $\leq 10$ )	8 60 min Weekly	52.0	8
Freedland et al., (2009)	Manualised CBT Involved problem solving, behavioural activation, cognitive techniques, and relapse-prevention. Provided by two clinical social workers and a counselling psychologist.	Out patient Individual	12 50-60 min Weekly	4.9	6
Foley et al., (2010).	MBCT. Involved mindful meditation practice with psycho-education on the relationship between thinking and mood with a specific focus on the role of cognitions in maintaining depressed mood. Delivered by one MBCT trained clinician.	Out patient Group (8-12)	8 120 min weekly	6.9	3
Hynninen et al., (2010)	Manualised CBT Involved strategies to modify beliefs and change behavioural patterns that may maintain psychological and somatic symptoms. Delivered by Masters-level psychology student.	Out patient Group (4-6)	7 120 min Weekly	9.8	6
Dao et al., (2011)	Brief manualised CBT: Involved psycho-education, developing problems list, setting behavioural goals, develop cognitive strategies, and reviewing progress. Delivered by two clinical psychologists	Hospital Individual	4 60 min within 3 weeks	3.0	~ 0.5
Dobkin et al., (2011)	Manualised CBT. including behavioural activation, thought restructuring, exercise, relaxation training, sleep hygiene and worry control. Also, four individual caregiver educational sessions (30–45 minutes) to support consolidation of CBT intervention. delivered by three doctoral-level psychologists.	Out patient Individual	10 / 60-70 min / Weekly	10.0	1

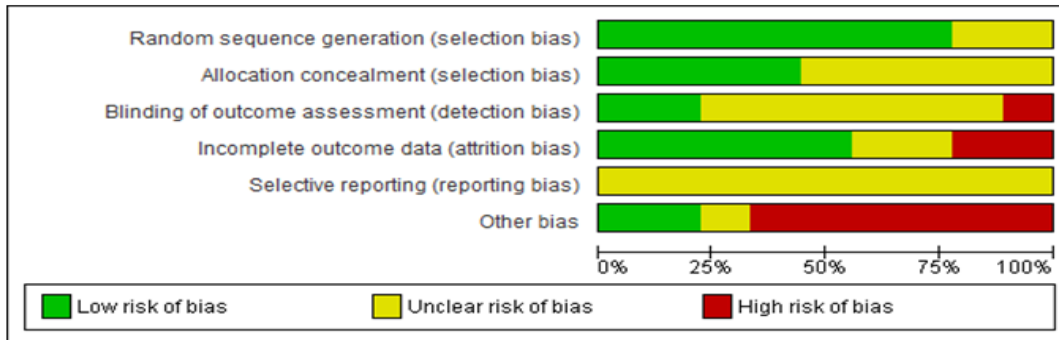
KEY: CBT = Cognitive Behavioural therapy. PST = Problem Solving Therapy. MBCT = Mindfulness based cognitive therapy.



Table 3: Characteristics of Included Studies: Outcomes

Authors (Date)	Assessment tool	Outcomes								
		Pre-			Post			Follow up		
		CBT	Control	Comp.	CBT	Control	Comp.	CBT	Control	Comp.
Lincoln & Flannaghan (2003)	BDI	17*	18*	15*	15.21 (10.1)	16.32 (8.39)	14.33 (8.42)	14.29 (7.98)	15.28 (8.7)	13.66 (9.46)
Gellis et al., (2008)	GDS-15	15.25 (6.1)	15.3 (6.4)		8.11 (4.3)	13.64 (5.6)		9.82 (4.57)	20.14 (3.48)	
	HDRS	20.31 (4.26)	20.72 (4.53)		10.06 (3.68)	20.84 (3.96)		8.48 (4.5)	13.53 (5.7)	
Koertge et al., (2008)	BDI	11.2 (6.2)	10.7 (7.1)		9.8 (6)	9.5 (6.8)		8.9 (7.3)	8.9 (6.8)	
Kunik et al., (2008)	BDI-II	23.44 (12.49)		21.12 (12.09)	14.19 (13.69)		14.54 (13.47)	15.47 (14.43)		15.04 (14)
Freedland et al., (2009)†	BDI	22.3† (8.32)	20.8† (8.85)	23.7 † (8.22)	5.4 † (8.32)	13.8† (8.85)	5.5† (6.4)	10.3† (6.32)	7.7† (6.48)	
	HDRS	19.3† (6.4)	18.5† (6.32)	20.8† (6.48)	5.5† (6.4)	10.7† (6.3)	6.7† (8.32)	12.9† (8.85)	9.9† (9.07)	
Foley et al., (2010)	HDRS	16.02 (7.28)	14.38 (8.12)		6.26 (5.43)	10.27 (6.93)		5.76 (5.3)		
Hynninen et al., (2010)	BDI-II	20.7 (8.6)	20.5 (9.7)		14.8 (7.8)	19.5 (9.4)		13.4 (5.9)	19.7 (8.9)	
Dao et al., (2011)	BDI-II	23 (6.6)	22.4 (6.2)		15.9 (5.1)	23.4 (11.4)		19.2 (6.7)	22.5 (10.7)	
Dobkin et al., (2011)	BDI	19.18 (7.47)	19.05 (7.37)		9.74 (7.4)	17.45 (7.17)		11.18 (7.58)	16.2 (7.39)	
	HDRS	20.93 (4.56)	19.38 (4.56)		13.58 (4.72)	19.33 (4.55)		14.52 (4.75)	19.31 (4.63)	

KEY: \* = median not mean; Standard Deviation not reported. BDI = Beck's depression Inventory. CBT = Cognitive Behavioural Therapy. Comp = Comparison Intervention. HDRS = Hamilton Depression Rating Scale. GDS = Glasgow Depression Scale. Standard Deviation shown in brackets. †Data for Freedland et al., (2009) is the covariate-adjusted least-squares mean. Reported standard deviation shown in brackets was from standard error data.



**Figure 2. Risk of bias graph: review raters’ judgements about each risk of bias item presented as percentages across all included studies**

**5.3.4. Effectiveness of CBT compared with treatment as usual (TAU) / Wait-list (WL) controls at end of treatment**

Data were available for 8 studies. self-rated measures were used when possible: BDI (Dobkin et al., 2011; Freedland et al., 2009; Koertge et al., 2008; Lincoln & Flannaghan, 2003), BDI-II (Dao et al., 2011; Hynninen et al., 2010) and GDS (Gellis et al., 2008). Clinician-rated outcome (HDRS) was used for Foley et al., (2010) in the absence of self-report measures.

Table 4 shows a medium effect favouring CBT over TAU / WL controls (pooled SMD = 0.63, 95% CI, 0.29 to 0.97) which was statistically significant (p = 0.0003). There was significant heterogeneity ( $X^2 = 37.49$ ,  $df = 7$  (p = <0.00001), with the  $I^2$  statistic indicating that between study heterogeneity accounted for 81% of variance in the effect size.

**Table 4. Forest plot showing effectiveness of CBT compared with TAU/WL control at end of treatment**

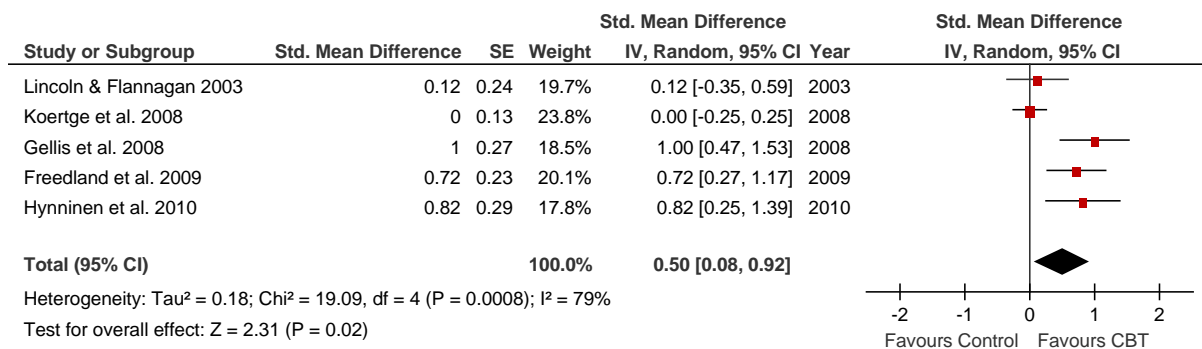
Study or Subgroup	Std. Mean Difference	SE	Weight	Std. Mean Difference		Year
				IV, Random, 95% CI	IV, Random, 95% CI	
Lincoln & Flannagan 2003	0.12	0.23	12.4%	0.12 [-0.33, 0.57]		2003
Gellis et al. 2008	1.09	0.27	11.5%	1.09 [0.56, 1.62]		2008
Koertge et al. 2008	-0.05	0.13	14.5%	-0.05 [-0.30, 0.20]		2008
Freedland et al. 2009	0.97	0.24	12.2%	0.97 [0.50, 1.44]		2009
Hynninen et al. 2010	0.53	0.29	11.0%	0.53 [-0.04, 1.10]		2010
Foley et al. 2010	0.64	0.19	13.3%	0.64 [0.27, 1.01]		2010
Dao et al. 2011	0.83	0.21	12.9%	0.83 [0.42, 1.24]		2011
Dobkin et al. 2011	1.05	0.24	12.2%	1.05 [0.58, 1.52]		2011
<b>Total (95% CI)</b>			<b>100.0%</b>	<b>0.63 [0.29, 0.97]</b>		

Heterogeneity: Tau<sup>2</sup> = 0.19; Chi<sup>2</sup> = 37.49,  $df = 7$  (P < 0.00001);  $I^2 = 81%$   
 Test for overall effect: Z = 3.59 (P = 0.0003)

### 5.3.5. Effectiveness of CBT compared with TAU/WL control at follow up

Data were available for 5 studies. (Freedland et al., 2009; Gellis et al., 2008; Hynninen et al., 2010; Koertge et al., 2008; Lincoln & Flannaghan, 2003). Follow up periods ranged from 3 months to 1-2 years (see table 2) with a mean follow-up period of 7.3 months. Table 5 shows a significant medium effect size favouring CBT over control at follow up (SMD = 0.5, 95% CI, 0.08 to 0.92) Heterogeneity was significant ( $X^2 = 19.09$ ,  $df = 4$ ,  $p = <0.0008$ ,  $I^2 = 79\%$ ).

**Table 5. Forest plot showing effectiveness of CBT compared with TAU/WL control at follow up**

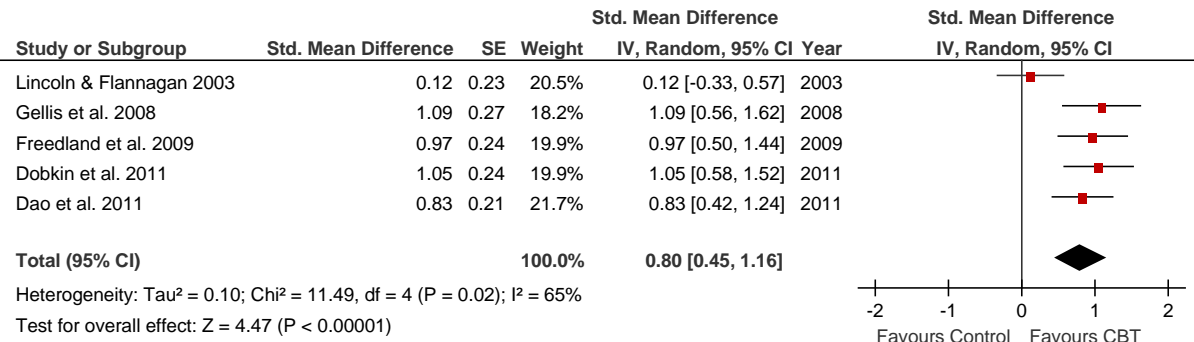


### 5.3.6. Sensitivity Analyses

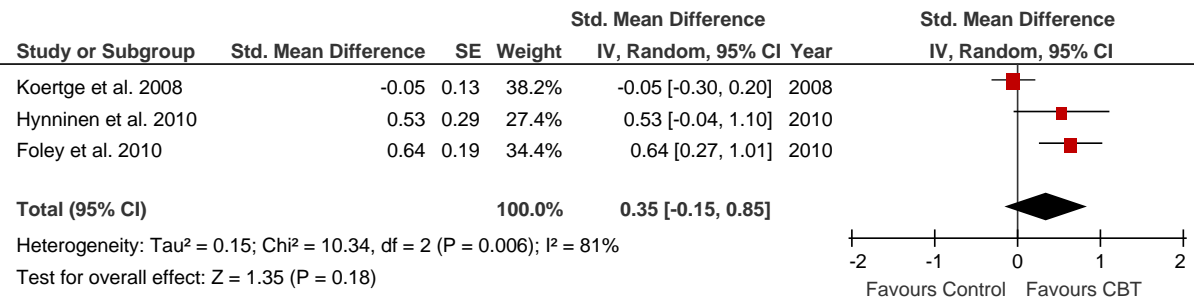
As specified a priori, the potential that differing modality of delivery and differing depression severity may introduce heterogeneity was explored with sensitivity analyses.

#### 5.3.6.1. Effectiveness of individual CBT intervention compared with group CBT

The analysis was re-run for group and individual studies separately. Only including studies which compared Individual CBT with TAU/WL control (Table 6) showed a large, statistically significant summary effect size (SMD) of 0.80 (95 % CI, 0.45 to 1.16,  $p = 0.00001$ ). Heterogeneity was significant ( $X^2 = 11.49$ ,  $df = 4$ ,  $p = <0.02$ ) with the  $I^2$  statistic indicating that 65% of variance in the effect size could be attributed to heterogeneity between included studies. Further sensitivity analysis to explore sources of heterogeneity was undertaken as recommended by the Cochrane collaboration (Higgins & Green 2011). Removing Lincoln & Flannaghan (2003) data yielded a highly significant summary effect size of 0.97 (95% CI, -0.09 to 0.57,  $p = 0.00001$ ) with no significant heterogeneity ( $X^2 = 0.75$ ,  $df = 3$ ,  $p = 0.86$ ,  $I^2 = 0\%$ ).

**Table 6. Forest plot showing effectiveness of individual CBT intervention compared with control**

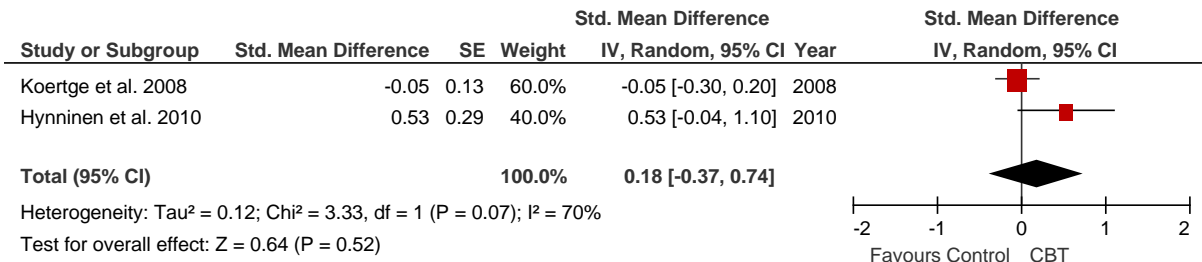
Referring to table 8 we can see a small, non-significant effect ( $p = 0.18$ ) favouring group CBT over TAU / WL control (SMD = 0.35, 95% CI, -0.15 to 0.85). Heterogeneity was significant ( $X^2 = 10.34$ ,  $df = 2$ ,  $p = <0.006$ ) with the  $I^2$  statistic indicating that 81% of variance in the effect size could be attributed to heterogeneity between included studies.

**Table 7. Forest plot showing effectiveness of group CBT intervention compared with control**

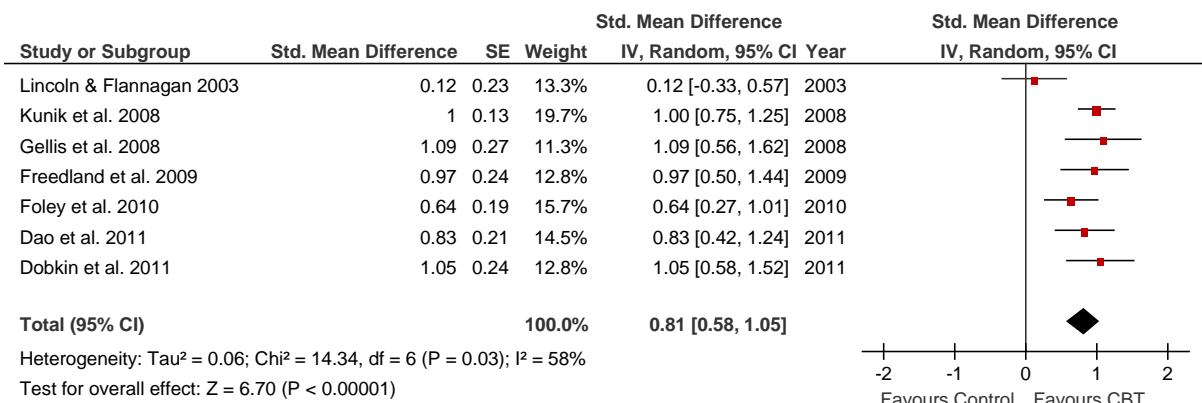
### 5.3.6.2. Outcomes for mild depression compared with moderate to severe depression

Only two studies had mean pre-treatment depression scores which fell within the 'mild' range (Hynninen et al., 2010; Koertge et al., 2008). Referring to table 8 we can see that only including these studies revealed no significant difference between CBT and control interventions (SMD = 0.18, 95% CI, -0.37 to 0.74,  $p = 0.52$ ). The remaining seven studies included samples with moderate to severe pre-treatment depression scores. Table 9 shows that these studies yielded a large, highly significant summary effect size (SMD = 0.81, 95% CI, 0.58 to 1.05,  $p = 0.00001$ ) with moderate heterogeneity ( $X^2 = 14.34$ ,  $df = 6$ ,  $p = 0.03$ ,  $I^2 = 58\%$ ).

**Table 8. Forest plot showing effectiveness of CBT intervention compared with controls for studies with mean depression severity scores indicative of mild depression**



**Table 9. Forest plot showing effectiveness of CBT intervention compared with controls for studies with mean depression severity scores indicative of moderate to severe depression**



### 5.3.7. Further Analyses

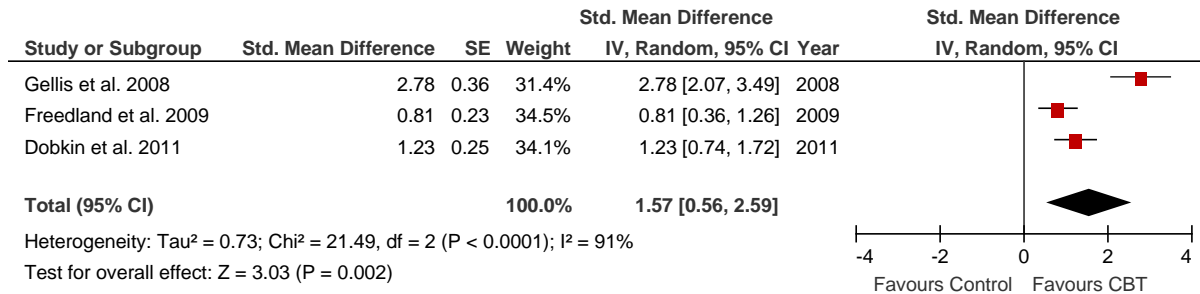
Outcome data yielded by clinician-rated measures and patient-rated measures were compared, as was the effectiveness of CBT when compared with 'active controls' as opposed to TAU/WL controls.

#### 5.3.7.1. Comparison of clinician-rated and self-rated measures of outcome

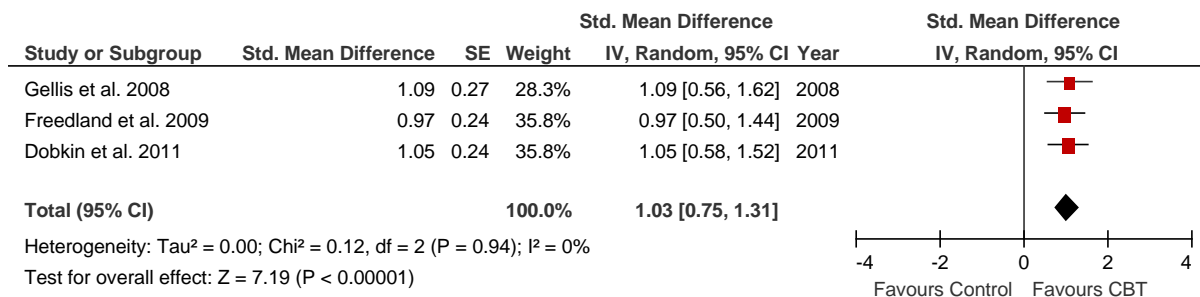
Referring to Tables 10 and 11 we can see that three studies (Dobkin et al., 2011; Freedland et al., 2009; Gellis et al., 2008) used both patient-rated and clinician-rated outcome measures and so were suitable for a comparative analysis. At end of treatment clinician-rated measures yielded a larger effect size, with a SMD of 1.57 (95% CI, 0.56 to 2.59) as compared with 1.03 (95% CI, 0.75 to 1.31). Both were significant (clinician-rated  $p = 0.002$ , patient rated  $p = 0.0001$ ) however the clinician-rated measures showed significantly greater heterogeneity ( $X^2 = 21.49$ ,  $df = 2$ ,  $p = 0.0001$ ,  $I^2 = 91\%$ ) than patient-rated measures ( $X^2 = 0.12$ ,  $df = 2$ ,  $p = 0.94$ ,  $I^2 = 0\%$ ). At follow up clinician-rated measures showed a reduced SMD of 1.22 (95% CI, 0.08 to 2.36,  $p = 0.04$ ) and increased heterogeneity ( $X^2 = 29.89$ ,  $df = 2$ ,  $p = 0.00001$ ,  $I^2 = 93\%$ ). Patient-rated outcome measures at follow up also showed a reduced

SMD (0.64, 95 % CI 0.27 to 1.01,  $p = 0.0007$ ) with heterogeneity increasing, although not reaching significance ( $X^2 = 3.75$ ,  $df = 2$ ,  $p = 0.15$ ,  $I^2 = 47\%$ ).

**Table 10. Forest plot showing effectiveness of CBT intervention compared with controls as measured by clinician-rated measures (only including those studies which employed both patient-rated and clinician-rated measure)**

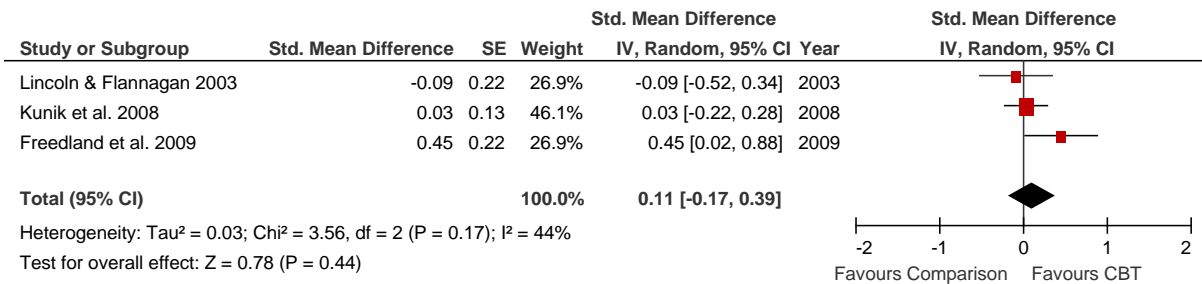


**Table 11. Forest plot showing effectiveness of CBT intervention compared with controls as measured by patient-rated measures (only including those studies which employed both patient-rated and clinician-rated measures)**



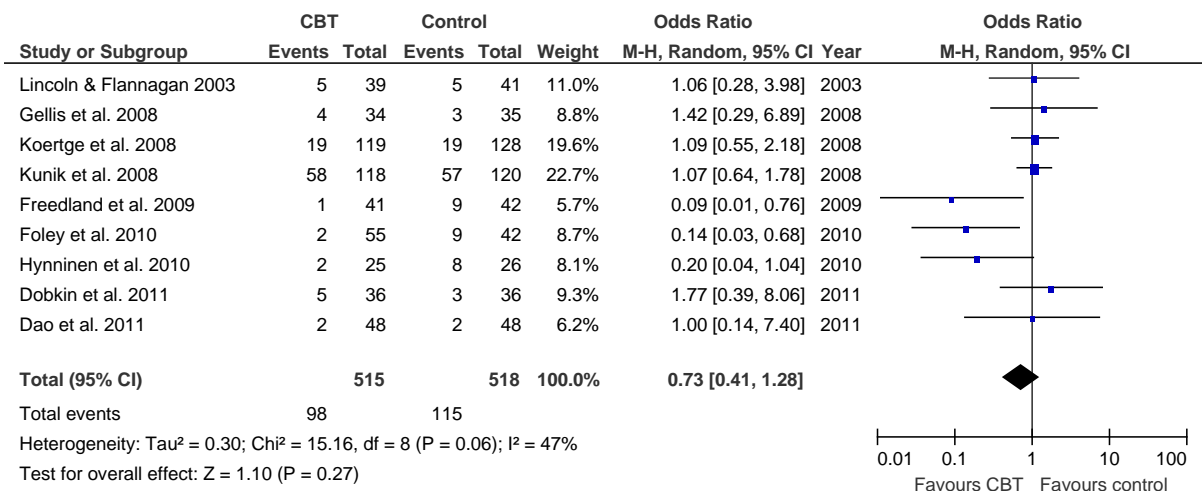
### 5.3.7.2. Effectiveness of CBT compared with 'active controls'

Only three studies compared the efficacy of CBT with alternative interventions. Freedland et al. (2009) compared CBT with stress management, Kunik et al. (2008) with COPD education, and Lincoln & Flannaghan (2003) with an attention placebo. Table 12 shows a small, non-significant effect favouring group CBT over active control (SMD = 0.11, 95 % CI, -0.17 to 0.39,  $p = 0.44$ ). Heterogeneity was not significant ( $X^2 = 3.56$ ,  $df = 2$ ,  $p = 0.17$ ) with the  $I^2$  statistic indicating that 44% of variance in the effect size could be attributed to heterogeneity between included studies. Heterogeneity reduced at follow up but remained non significant ( $X^2 = 2.42$ ,  $df = 2$   $p = 0.30$ ,  $I^2 = 17\%$ ) as did the summary effect size (SMD = 0.08, 95 % CI, -0.18 to 0.33,  $p = 0.56$ ).

**Table 12. Forest plot showing Effectiveness of CBT compared with active controls**

### 5.3.8. Analysis of drop out

Mean attrition across comparisons of CBT to TAU / WL control was 20%, with two studies showing particularly high attrition (Koertge et al., 2008; Kunik et al., 2008). Eighty four per cent of the 7316 patients screened across all studies were excluded before randomisation. All studies provided data regarding drop out between baseline and treatment completion for CBT and control groups. Referring to table 13 we can see the pooled odds ratio was 0.73 (95% CI, 0.41 to 1.28] with patients more likely to drop out of control conditions, which was not statistically significant ( $p = 0.27$ ).

**Table 13. Forest plot showing pooled odds ratio of drop-out for CBT groups and control groups**

### 5.3.9. Tests for publication bias

Due to the small number of included studies, visual inspection of funnel plot was considered an unreliable method of assessing possible publication bias (Terrin et al., 2005). (The funnel plot of summary effect size against standard error is included as Appendix 15). Fail-safe N data were calculated according to methods outlined by Rosenthal (1979) and Rosenberg (2005). Rosenthal's fail-safe N (Rosenthal 1979) indicated 170 unpublished studies with an effect size of zero would be

required to render the population effect size non-significant. This figure exceeds  $5n+10$ , indicating the results can be considered robust to the effects of publication bias (Rosenthal 1991). A still robust fail-safe  $N$  of 119 was found applying Rosenberg's (2005) more conservative methods which employ a fixed-effect model and include procedures to weight studies.

## 5.4. Discussion

### 5.4.1. Summary of main findings

This study found that CBT showed a medium effect ( $SMD = 0.63$ ) in reducing depressive symptoms in older adults with underlying physical illnesses when compared with treatment as usual or waiting list controls and that these benefits were largely sustained at follow up ( $SMD = 0.50$ ). Previous meta-analyses examining CBT for late-life depression, which have not explicitly focused on older people with co-morbid physical illnesses, have reported higher summary effect sizes for the impact of CBT on depressive symptoms as compared with controls: Pincuart et al. (2007),  $d = 1.12$ ; Cuijpers et al. (2006),  $d = 0.70$ ; Pincuart et al. (2006),  $d = 0.88$ . However, these studies employed Cohen's  $d$  as a measure of the SMD rather than the more conservative Hedges'  $g$  used in this study, making comparison misleading. In fact, Pincuart & Sorensen (2001), employing Hedges'  $g$ , using self-rated outcome measures and not focusing explicitly on older adults with physical illness found a very similar effect size ( $g = 0.64$ ) for the effect of CBT on depressive symptoms in late-life depression. Although these results appear consistent, it is not possible to conclude from this that physical co-morbidity has no impact on treatment outcomes. Samples of older adults in previous meta-analyses have tended to include significant degrees of co-morbidity, and a previous study, undertaking moderator analyses and including a much larger sample (Pincuart et al. 2007) found physical co-morbidities were associated with reduced treatment effect. Nevertheless results from the current study indicate that CBT is more effective than treatment as usual or no intervention in reducing depressive symptoms in older adults with underlying physical illnesses.

### 5.4.2. Exploration of heterogeneity

In this study the summary effect size (SMD) found for CBT in reducing depressive symptoms in older adults with underlying physical illnesses showed significant heterogeneity. Sensitivity analyses showed that only including studies that employed individual rather than group CBT reduced heterogeneity and increased the summary effect size. These findings might suggest individual psychotherapy is more effective than group approaches in treating late-life depression (Cuijpers, et al. 2008b) however, this conclusion is not supported by a number of other previous



studies (Bohlmeijer et al. 2003; Cuijpers, 1998; Cuijpers et al. 2006; Pincus et al. 2007; Scogin & McElreath, 1994, Cuijpers et al. 2008a) and sensitivity analyses designed to explore heterogeneity should not be interpreted as yielding data regarding moderators of treatment.

Further sensitivity analysis explored the role of depression severity in potentially explaining heterogeneity of effect sizes between studies. A distinction was made between mild depression and moderate to severe depression as this is the threshold at which the National Institute for Health and Clinical Excellence (NICE, 2009) recommend the use of antidepressant therapy. In studies which included patients with mean depression scores falling in the mild range (Hynninen et al., 2010; Koertge et al., 2008) CBT did not show greater efficacy than controls in reducing depression symptoms (Table 8). However, only including studies with mean depression scores falling in the moderate to severe ranges, revealed a large summary effect size for the efficacy of CBT in reducing depressive symptoms (Table 9). This is consistent with previous findings that elevated depression symptoms may be associated with larger effect sizes for psychotherapeutic interventions (Beltman et al., 2010; Bohlmeijer et al., 2003). However this interpretation must be considered in light of the fact that, due to the small number of studies, the current analysis was underpowered to detect sub-group differences. In addition, a number of previous meta-analyses have not found such a relationship (Huxtable, Section 2; Cuijpers et al., 2008a) and using cut-off scores on validated outcome measures to assess depression severity is potentially problematic: varying cut-offs have been proposed depending on the measures used and the nature of an individual's underlying physical health condition (Strober & Arnett 2009) and scores on depression ratings cannot be considered equivalent to diagnosis using standardised assessment schedules. Although sensitivity analyses seem to point to a differential efficacy of CBT according to depression severity and treatment modality in older adults with underlying physical illnesses, further analysis including sufficient studies to allow formal sub-group analysis would be required to confirm these initial findings.

#### **5.4.3. Further analyses**

This study was able to analyse three studies which compared CBT with 'active controls': Freedland et al. (2009) employed a stress management condition; Kunik et al., (2008) a COPD education programme; Lincoln & Flannaghan (2003) utilised an attention placebo. Although these comparative interventions varied considerably, and including an attention placebo with alternative treatments in the analysis could be considered potentially problematic, the heterogeneity of this comparison was not found to be significant. No significant difference was found between CBT and these active controls. This finding is consistent with previous meta-analyses which have not found a consistent difference between the effectiveness of CBT and alternative psychotherapeutic approaches

for late life depression (Huxtable, Section 2). Attrition was explored as a proxy for treatment acceptability. Patients were no more likely to drop out of CBT interventions than controls. This indicates that CBT is potentially an acceptable form of treatment in this group of patients, however as comparisons were with TAU / WL controls, no conclusions regarding the relative acceptability of differing approaches could be made.

Three of the included studies used both clinician-rated and self-report measures of depressive symptoms. The majority of previous meta-analyses examining psychotherapeutic interventions for late-life depression have found clinician-rated measures to yield higher effect sizes than self-report measures within the same studies (Huxtable, Section 2) and a meta-analysis exploring this question explicitly, found this differential to be statistically significant (Cuijpers, et al., 2010). Results from the current study were in line with these findings, with clinician-rated measures yielding a larger effect size than patient-rated measures at end of treatment (SMD of 1.57 as compared with 1.03).

#### **5.4.4. Methodological considerations**

Considering the potential heterogeneity of the patient group examined, a random effects model was used. It was assumed that studies were drawn from research domains that potentially differed systematically (in that they explored varying physical illnesses) and that variation in effect sizes between studies may reflect not only random error within studies, but also true variation in effect size between studies. In an attempt to reduce this risk of ‘comparing apples and oranges’ (Eysenck, 1984), this study employed a well-delineated and specific a-priori search strategy. However this resulted in a relatively narrow focus on a small selection of studies which potentially limited the degree to which the results could be understood as representative of a broader and heterogeneous research domain (Sharpe, 1997).

Grouping wait-list controls and treatment as usual control together for the purposes of comparative analysis also must be considered critically. There is a risk those in wait-list groups may seek alternative, uncontrolled, interventions out with the study criteria. Cuijpers et al. (2008a) found that effect sizes were reduced in studies using treatment as usual as opposed to waiting list controls and it is likely patients’ expectations of improvement will differ between these groups. Despite these risks, it was judged that combining these two types of control was useful to the extent that it facilitated an exploratory understanding of the relative efficacy of CBT interventions when compared with groups receiving no psychotherapeutic intervention.

Studies were included in this meta-analysis which evidenced core cognitive and behavioural interventions focused specifically on reduction of depressive symptoms. Studies employing problems

solving approaches were included if they met this very specific criteria. However, the exclusion of studies employing non-specific CBT or CBT focused on anxiety symptoms significantly reduced the number of eligible studies. In light of evidence that anxiety and mood disorders are frequently co-morbid (Wolitzky-Taylor et al., 2010) and that in studies excluded from the current analysis, treatment for anxiety frequently positively impacted on depression, (Blumenthal et al., 2005; Evers et al., 2002; Kunik et al., 2001; Sharpe et al., 2003; Sharpe et al., 2001) it could be argued that a meta-analysis exploring both conditions may have yielded more generalisable findings.

#### 5.4.5. Limitations and risk of bias

The quality of included papers was not optimum, with four studies not undertaking suitable power analyses (Dao et al., 2011; Foley et al., 2010; Gellis et al., 2007; Koertge et al., 2008) and the majority of studies failing to either adequately blind outcome assessors or undertake robust tests for treatment fidelity. High attrition and high exclusion was seen across the included studies and reflects the fact that barriers to treatment are particularly significant in this patient group. These factors, considered in light of the variation in study design with regards length of therapy, training of clinicians and intensity of intervention mean generalising from these results must be undertaken cautiously. Particularly as the study only captured a limited range of co-morbid health conditions and examined a predominantly young-old patient sample.

This study was not able to shed light on how depression may present differently in older-adults with underlying physical health conditions (Moorey & Steiner 2007) or explore moderating and mediating factors. In addition, it was not possible to control for the stage of the underlying illness, analyse results according to a measure of overall disability or establish adequate conclusions with regards longer-term outcomes.

It is possible that publication bias may have impacted overall results (Cuijpers, Smit, Bohlmeijer, Hollon, & Andersson, 2010) although the systematic search sought to identify unpublished studies, and subsequent tests for publication bias were found to be robust. A previous study examining CBT for depression in adults with a somatic disease (Beltman et al., 2010) failed to find significant effects for the impact of CBT on depressive symptoms, once studies without intent to treat designs were removed from the analysis. By way of contrast, although study quality in this analysis was less than optimum, eight out of the nine studies included intent to treat analyses or used 95% completer data, indicating that the finding that CBT shows a moderately large impact on depressive symptom for older adults with an underlying physical illness, can be considered robust to the impact of attrition bias.

#### 5.4.6. Clinical implications

Older adults receive poorer care than their younger counterparts (Bartels, 2002) with depression under-diagnosed and under-treated (Age Concern, 2008). Functional impairment and medical co-morbidity increase the risk of depression for older adults (Blazer 2003) and there is a further risk that physical co-morbidity may confound the diagnosis of depression (Charlson & Peterson 2002). Cognitive behavioural therapy has the strongest evidence base amongst psychological therapies for late-life depression (Bartels et al., 2003; Gatz, 2007; Laidlaw, 2001; Scogin et al., 2005). However, prior to this study, no meta-analysis had systematically examined the efficacy of CBT in reducing depressive symptoms in older patients with physical illnesses. A previous large scale study found that co-morbid physical illnesses did not negatively impact on the efficacy of collaborative care interventions for depression in this age group (Harpole et al., 2005). In light of the fact that, independent of medical co-morbidity, depression and depressive symptoms are associated with significantly higher health-care costs (Katon et al. 2003), the finding that CBT is effective in reducing symptoms of depression in this group of patients means there is a strong clinical argument for ensuring older adults experiencing depressive symptoms in the context of physical illness, receive appropriate psychotherapeutic assessment and treatment with CBT considered a first-choice treatment option.

#### 5.4.7. Conclusions

Findings from this meta-analysis indicate CBT is more effective than treatment as usual or waiting list controls in reducing depression symptoms in older adults with underlying physical illnesses. Results suggest that individual rather than group approaches may be associated with greater efficacy, although further studies are required to explore this hypothesis and care should be taken generalising results from the current study due to the small number of included studies. Further studies need to be undertaken to develop an understanding of what factors may moderate or mediate the effectiveness of CBT in this group of patients particularly as comparisons with alternative treatments did not find an advantage for CBT. Future studies should include standardised measures of illness burden to facilitate a better understanding of the interaction between psychological therapy and differing underlying somatic complaints. Future research should also focus particularly on the physically ill older-old whose needs have been inadequately addressed with good quality randomised controlled trials to date.

Word Count: 6194

#### 5.4.8. Highlights

(3-5 bullet points, maximum 85 characters, including spaces, per bullet point: See Appendix 1)

- CBT reduces depression symptoms in older adults with physical illnesses
- Small number of studies means generalising from results should be done with caution
- Patient-rated measures yielded smaller effect sizes than clinician-rated measures.
- There is a need for more studies of psychotherapy efficacy with the older-old.

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**SECTION 6: Additional Methodology**

Outlined in this section are the exact methods undertaken in the meta-analysis described in Section 4, with full details to facilitate replication.

## 6. Additional Methodology

### 6.1. Calculation of the standardized mean difference

All the included studies were randomized controlled trials, employing independent groups. A variety of validated outcome measures were used: BDI (Beck et al., 1961); BDI-II (Beck et al., 1996); GDS-15 (van Marwijk et al., 1995); HDRS (Hamilton, 1967). To facilitate comparison of studies using different outcome measures, the standardised mean difference ( $\delta$ ) was calculated for each study according to methods outlined in Borenstein *et al.*, (2009).

The index  $\delta$ , denotes the effect size parameter. Cohen's 'd' (Cohen, 1987) on the other hand, refers to a specific sample estimate of this parameter. To calculate  $\delta$ , the mean from the control group ( $X_2$ ) was subtracted from the mean of the intervention group ( $X_1$ ) and divided by the within-groups standard deviation, pooled across groups. All calculations were undertaken in Microsoft Excel.

$$d = \frac{\bar{X}_1 - \bar{X}_2}{S_{within}} \quad (1.0)$$

This treatment makes the common parametric assumption that both independent groups share a common true (population) standard deviation. However, assuming the true (population) standard deviation is the same, it is nonetheless likely that sample estimates of standard deviation will not be identical due to sampling error. Pooling them therefore gives us more data and yields a more accurate estimation of their common value.

To calculate the pooled within-group standard deviation the following equation was used:

$$S_{within} = \sqrt{\frac{(n_1 - 1)S_1^2 + (n_2 - 1)S_2^2}{n_1 + n_2 - 2}} \quad (1.1)$$

Here,  $n_1$  and  $n_2$  are the sample sizes in the two groups and  $S_1$  and  $S_2$  are the standard deviations in the two groups.

The variance of  $d$  was calculated.

$$V_{d = \frac{n_1 + n_2}{n_1 n_2} + \frac{d^2}{2(n_1 + n_2)}} \quad (1.2)$$

Here the first term ( $\frac{n_1 + n_2}{n_1 n_2}$ ) reflects the uncertainty in the estimate of the means difference, i.e. the numerator in (1.0). The second term ( $\frac{d^2}{2(n_1 + n_2)}$ ) reflects the uncertainty in the estimate of  $S_{within}$ .

The standard error of  $d$  was calculated (the square root of the variance of  $d$ ).

$$SE_{d = \sqrt{V_d}}$$

(1.3)

Cohen's  $d$  has a tendency to overestimate the population parameter effect size ( $\delta$ ) when small samples are used (Borenstein *et al.*, 2009). Converting to Hedges'  $g$  (Hedges, 1981) corrects for this, and is done by employing a correction factor:  $J$ .

The correction factor  $J$  was calculated.

$$J = 1 - \frac{3}{4df - 1}$$
(1.4)

Here, the degrees of freedom ( $df$ ) is the  $df$  used to estimate  $S_{within}$ . For two independent groups (as in this study)  $df$  is the denominator in (1.1) i.e.  $n_1 + n_2 - 2$ :

$$J = 1 - \frac{3}{4(n_1 + n_2 - 2) - 1}$$
(1.5)

Cohen's  $d$  was then converted to Hedges'  $g$ .

$$g = J \times d$$
(1.6)

The variance of  $d$  was then converted to the variance of  $g$ .

$$V_g = J^2 \times V_d$$
(1.7)

The Standard Error of  $g$  was then calculated.

$$SE_g = \sqrt{V_b}$$
(1.8)

Hedges'  $g$ , with variance and standard error data were calculated for each study. Separate calculations were undertaken according to both self-rated and clinician-rated measures where both were available. Separate calculations were made for each comparison in studies which included an active treatment condition in addition to TAU/WL control: CBT ( $X_1$ ) was compared with treatment as usual ( $X_2$ ). And then CBT ( $X_1$ ) was compared with alternative treatment ( $X_2$ ).

$$d = \frac{\bar{X}_1 - \bar{X}_2}{S_{within}}$$

Data were then entered into the Cochrane Collaboration Review Manager Software (RevMan 5.1, 2011) and synthesized meta-analytically.

## 6.2. Calculation of the odds ratio

For each study, a 2x2 table was generated with CBT and control conditions and numbers of patients randomised to, and dropping out of each condition.

	<i>n</i> drop-out	<i>n</i> randomised
CBT	A	B
Control	C	D

The odds ratio for each study was calculated:

$$\text{Odds Ratio} = \frac{AD}{BC}$$

Data from the cells was entered into RevMan 5.1 (RevMan 5.1, 2011) using a random effects model. Odds ratios for each study were cross checked and a summary odds ratio calculated.

## 6.3. Calculation of fail-safe N: Assessing publication bias.

Rosenthal's fail-safe N (Rosenthal, 1979) calculates the number of additional studies ( $N_R$ ) with a mean null result that would be necessary reduce the overall significance of the mean  $Z$  score to a desired significance level (usually  $\alpha = 0.05$ ). Data were calculated according to the below equation where  $N$  is the number of studies,  $Z(\pi_i)$  is the  $Z$  scores for individual significance values and  $Z_{\alpha}$ , the  $Z$  score (one-tailed) associated with the desired  $\alpha$ .

$$N_{R} = \frac{[\sum Z(\pi_i)]^2}{Z_{\alpha}^2}$$

An on-line fail-safe-N calculator described by Rosenberg (2005) was used to calculate Rosenthal's fail-safe N according to the above equation (<http://www.rosenberglab.net/software.php#failsafe>). The effect size ( $g$ ) and variance for each study was entered into the programme. Data from CBT versus control (WL or TAU) at end of treatment, assessed by patient-rated depression measures were used for all studies except for Kunik *et al.*, (2008) where TAU or WL control comparison was not available and so data for comparison to an alternative treatment (COPD education) was used.

As Rosenthal's (1979) methods tend to over-estimate the number of studies required to nullify observed results, Rosenberg's (2005) methods were also applied. These differ in that each included study is weighted by the inverse of its variance. A fixed-effect model was employed. Detailed methods are outlined in Rosenberg (2005).

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Appendix 1: Guide to Authors:  
Clinical Psychology Review





# CLINICAL PSYCHOLOGY REVIEW

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**D.J. Hansen**, University of Nebraska at Lincoln, Lincoln, NE, USA

**M. Harrow**, University of Illinois College of Medicine, Chicago, IL, USA

**R. Heinssen**, National Institute of Mental Health (NIMH), Bethesda, MD, USA

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**R. Levin**, Albert Einstein College of Medicine, Bronx, NY, USA

**L. Mullins**, Oklahoma State University, Stillwater, OK, USA

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**J.T. Nigg**, Oregon Health and Science University (OHSU), Portland, OR, USA

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**N. Singh**, Chesterfield, USA

**S. Taylor**, University of British Columbia, Vancouver, BC, Canada

**D. Velligan**, UTHSCA, San Antonio, TX, USA

**B. Wampold**, University of Wisconsin at Madison, Madison, WI, USA

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**T. Wykes**, University of London, London, UK

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## Appendix 2: Full Search Strategy (Systematic Review).

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#	Query	Limiters/Expanders	Last Run Via	Results
S7	S1 and S2 and S3 and S4	Search modes - Boolean/Phrase	Interface - EBSCOhost Search Screen - Basic Search Database - CINAHL Plus with Full Text;MEDLINE with Full Text;PsycINFO;Psychology and Behavioral Sciences Collection;Health Business Elite;Biomedical Reference Collection: Comprehensive;Library, Information Science & Technology Abstracts;eBook Collection (EBSCOhost);Nursing Reference Center	343
S6	S1 and S2 and S3	Search modes - Boolean/Phrase	Interface - EBSCOhost Search Screen - Basic Search Database - CINAHL Plus with Full Text;MEDLINE with Full Text;PsycINFO;Psychology and Behavioral Sciences Collection;Health Business Elite;Biomedical Reference Collection: Comprehensive;Library, Information Science & Technology Abstracts;eBook Collection (EBSCOhost);Nursing Reference Center	678
S5	empirical review OR evidence based review OR evidence-based review	Search modes - Boolean/Phrase	Interface - EBSCOhost Search Screen - Basic Search Database - CINAHL Plus with Full Text;MEDLINE with Full Text;PsycINFO;Psychology and Behavioral Sciences Collection;Health Business Elite;Biomedical Reference Collection: Comprehensive;Library, Information Science & Technology Abstracts;eBook Collection (EBSCOhost);Nursing Reference Center	2240
S4	psychotherap* OR psychosocial OR psychological OR cognitive OR behavioural OR psychodynamic OR non-medical OR non-pharmaceutical OR counselling OR inter-personal	Search modes - Boolean/Phrase	Interface - EBSCOhost Search Screen - Basic Search Database - CINAHL Plus with Full Text;MEDLINE with Full Text;PsycINFO;Psychology and Behavioral Sciences Collection;Health Business Elite;Biomedical Reference Collection: Comprehensive;Library, Information Science & Technology Abstracts;eBook Collection (EBSCOhost);Nursing Reference Center	2504135
S3	meta-analysis OR systematic review OR quantitative review	Search modes - Boolean/Phrase	Interface - EBSCOhost Search Screen - Basic Search Database - CINAHL Plus with Full Text;MEDLINE with Full Text;PsycINFO;Psychology and Behavioral Sciences Collection;Health Business Elite;Biomedical Reference Collection: Comprehensive;Library, Information Science & Technology Abstracts;eBook Collection (EBSCOhost);Nursing Reference Center	130045
S2	depress* OR dysthymi* OR mood	Search modes - Boolean/Phrase	Interface - EBSCOhost Search Screen - Basic Search Database - CINAHL Plus with Full Text;MEDLINE with Full Text;PsycINFO;Psychology and Behavioral Sciences Collection;Health Business Elite;Biomedical Reference Collection: Comprehensive;Library, Information Science & Technology Abstracts;eBook Collection (EBSCOhost);Nursing Reference Center	742534
S1	older adults OR older people OR geriat* OR elder* OR late life OR senior	Search modes - Boolean/Phrase	Interface - EBSCOhost Search Screen - Basic Search Database - CINAHL Plus with Full Text;MEDLINE with Full Text;PsycINFO;Psychology and Behavioral Sciences Collection;Health Business Elite;Biomedical Reference Collection: Comprehensive;Library, Information Science & Technology Abstracts;eBook Collection (EBSCOhost);Nursing Reference Center	643528

Appendix 3: Email correspondence with authors of selected studies (Systematic Review).

I am writing to you as the corresponding author for the meta-analysis: [Insert title of published meta-analysis]

I am currently undertaking a systematic review of meta-analyses which examine psychotherapeutic treatments for late-life depression. The study is being undertaken in part-fulfilment of a Doctorate in Clinical Psychology at Edinburgh University.

I am seeking to determine if you are aware of any ongoing or unpublished meta-analytic studies in this area that may inform my research question, and which I may not have been able to access via literature searches of published studies. I have outlined below a brief description of my research questions.

Research Questions:

1. What have meta-analyses revealed about which psychological interventions are effective in reducing depressive symptoms in older adults (>55years)?
2. What have meta-analyses revealed about predictors of treatment efficacy in late life depression?
3. What have meta-analyses revealed with regard to the role of moderating factors (age, gender, participant characteristics, severity and chronicity of disorder, treatment length, and therapeutic alliance etc..) in the successful treatment of late life depression?

The implications of these findings will be discussed within the broader literature which seeks to develop an empirical understanding of the specific factors which moderate psychotherapeutic efficacy.

If you are aware of any relevant unpublished research I would be very grateful if you could get in contact.

Yours Sincerely,

David Huxtable

#### Responses:

Author	Year	Contacted	Reply
Gorey, K. M., Cryns, A. G.	1991	Y	Y
Scogin, F., McElreath, L.	1994	Y	
Engels, G.I., Vermey, M., Cuijpers, P.	1997	Y	
Cuijpers, P.	1998	Y	
Pinquart, M., Sorensen, S.	2001	Y	Y
Bohlmeijer, E., Smit, F., Cuijpers, P.	2003	Y	
Cuijpers, P., van Straten, A., Smit, F.	2006	Y	
Pinquart, M., Duberstein, P.R., Lyness, J.M.	2006	Y	Y
Chin, A.	2007	Y	
Pinquart, M., Duberstein, P.R., Lyness, J.M	2007	Y	Y
Wilson, K., Mottram, P.G., Vassilas, C.	2008	Y	Y
Peng, X.D., Huang, C.Q., Chen, L.J., Lu, Z.C.,	2009	Y	
Samad, Z., Brealey, S., Gilbody, S.	2011	Y	Y
Krishna, M.,Jauhari, A., Lepping, P., Turner, J., Crossley, D., Krishnamoorthy, A.	2011	Y	Y

**Replies received:**

Martin Pinquart [pinquart@staff.uni-marburg.de]

Dear David,

I am aware of one unpublished study that is under review. Unfortunately, I cannot provide information on this study because I reviewed the manuscript and are not allowed to share informations until the study is published. Note that the paper you cited was not exclusively focused on depressed older adults. The following papers would be more relevant Pinquart, M., Duberstein, P., & Lyness, J.M. (2006). Treatments for later life depressive conditions: A meta-analytic comparison of pharmacotherapy and psychotherapy. *American Journal of Psychiatry*, 163, 1493-1501. Pinquart, M., Duberstein, P. & Lyness, J. (2007). Effects of psychotherapy and other behavioral interventions on clinically depressed older adults: A meta-analysis. *Aging and Mental Health*, 11, 645-657.

Yours,

Martin Pinquart

Von: "Huxtable David (NHS GRAMPIAN)" <davidhuxtable@nhs.net>  
 An: "Pinquart@staff.uni-marburg.de" <Pinquart@staff.uni-marburg.de>  
 Datum: Fri, 30 Mar 2012 13:15:23 +0100  
 Betreff: Enquiry re: How effective are psychotherapeutic and other psychosocial interventions with older adults?

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Simon Gilbody [simon.gilbody@york.ac.uk]

Dear David. All my reviews are in the public domain. Good luck with your doctorate. Simon

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Kevin Gorey [gorey@uwindsor.ca]

Attachments: SocWorkRes1998.pdf (561 KB) ResAging1992.pdf (2 MB) JGeriatPsych1990.pdf (807 KB)

Dear Mr. Huxtable:

Here are a few that may be at least of tangential use to you (perhaps not)--from my gerontological research days. And below is another that I no longer have copies of/nor access to. I do not have any such unpublished research in my "file drawers." Wheeler, J.A., Gorey, K.M., & Greenblatt, B. (1998). The beneficial effects of volunteering for older volunteers and the people they serve: A meta-analysis. *International Journal of Aging and Human Development*, 17, 69-79. Great luck with your dissertation. Love to read it when the time comes.

Kevin

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MURALI KRISHNA (BCUHB - Hergest) [MURALI.KRISHNA@wales.nhs.uk]

Telephone correspondence

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Wilson, Kenneth [K.C.M.Wilson@liverpool.ac.uk]

I refer you on to the Cochrane centre for depression, anxiety and neurosis for this enquiry as they are in contact with up to date literature searches  
 Prof Ken Wilson

Appendix 4: Summary results from each included study  
(Systematic Review).

Pinquart et al., (2007) *Effects of psychotherapy and other behavioral interventions on clinically depressed older adults: a meta-analysis.*

Analysis	Results									
	K	D	95% CI	T	Q	k	d	95% CI	t	Q
	Pre-post effects					Follow-up effects				
Reminiscence	8	1.00	0.73-1.27	7.18***	13.58	2	0.63	0.25-1.02	3.25**	0.37
CBT /Cognitive therapy/Behavioural therapy	35	1.06	0.87-1.26	10.54***	122.37***	10	0.79	0.45-1.13	4.54***	23.17**
Behavioural therapy	11	0.96	0.77-1.15	9.96***	11.74	5	0.69	0.41-0.97	4.80***	1.63
CBT	13	1.12	0.76-1.48	6.08***	40.22***	4	0.93	0.14-1.73	2.31*	20.59***
Cognitive therapy	10	1.06	0.64-1.47	5.04***	65.39***					
Psychodynamic therapy	3	0.76	0.31-1.21	3.29***	0.71					
Interpersonal Therapy	3	0.14	-0.16-0.43	0.91	0.35					
Overall: Self-rated depression	75	0.84	0.71-0.97	12.27***	175.62***					
Overall: Clinician rated depression	49	0.93	0.74-1.11	9.74***	161.28***					

Impact of Study Characteristic on treatment effects.	B	$\beta$	t
Control condition (active placebo/other)	-0.63	-0.53	-4.97***
Quality of Study (4=high; 0=low)	-0.22	-0.23	-2.25*
Major Depression vs Minor/Mixed/Dysthymia	-0.26	-0.22	-2.21*
Co-morbidity	-0.35	-0.22	-2.23*
Mean Age (linear)	0.18	1.56	0.55
Mean Age (quadratic)	0.00	-1.48	-0.56
Format (group vs other)	-0.18	-0.16	-1.59
Duration (sessions)	-0.01	-0.12	-1.17
Setting (inpatient vs other)	-0.19	-0.14	-1.29

Analysis of Drop-out			
Mean age (linear)	-0.61	-4.20	-0.91
Mean age (quadratic)	0.00	4.29	0.94
Group format (group=1; other=0)	0.49	0.36	2.45*
Number of sessions	0.01	0.25	1.99*
Control Condition	0.07	0.05	0.32
Quality of Study	-0.05	-0.04	-0.31
Depression severity,	0.08	0.05	0.29
Inpatient	-0.10	-0.06	-0.42
Comorbidity	-0.35	-0.15	-1.13

Analysis of Drop-Out	K	Odds	95%CI	t	Q
Sample composition at T1					
Major Depression	18	0.30	0.22-0.41	7.52***	29.91*
Major/Minor/Dysthymia	30	0.29	0.23-0.37	9.81***	45.28*
Minor/Dysthymia	2	0.11	0.02-0.52	2.77**	6.64*
Type of Intervention					
Cognitive Behavioural Therapy	25	0.29	0.21-0.39	7.84***	64.54***
Reminiscence	5	0.35	0.22-0.55	4.55***	2.97
Psychodynamic Therapy	3	0.29	0.16-0.53	4.05***	1.73
Interpersonal Therapy	2	0.18	0.09-0.34	5.29***	1.6

**Main Effect**

Self-rated depression improved by  $d = 0.84$  clinician-rated depression  $d = 0.93$ , both showing significant heterogeneity. CBT and reminiscence: large effect sizes. Psychodynamic therapy medium effect size.

**Sub-group analyses**

Weaker improvements of depressive symptoms were found in studies with active control group; physical co-morbidity; cognitively impaired patients; major depression (versus other mood disorders); studies of higher quality. Age, format, duration, outcome measure used and treatment setting did not show treatment effects. Higher dropout rates in group interventions and in longer interventions.

**KEY**

\* $p < 0.05$ ; \*\* $p < 0.01$ ; \*\*\* $p < 0.001$ . k = number of treated subsamples. d = effect size. 95% CI = 95% Confidence Interval of d. t = test of significance of effect size; Q = test of homogeneity of the effect size (significant values indicate heterogeneity) B/ $\beta$  = unstandardized/standardized regression coefficient.



**Chin (2007)** *Clinical effects of reminiscence therapy in older adults: A meta-analysis of controlled trials*

Analysis	Results				
	SMD	95% CI	X <sup>2</sup>	p	FsI
Reminiscence Vs Control. Impact of depression. (6 studies)	-0.9	-1.49 to -0.32	16.55	0.005	9
Reminiscence Vs Control (excluding studies with data in skewed distribution) (3 studies)	-1.39	-2.26 to -0.52			

**Main Effect**

Poor quality of studies, Small N, Significant heterogeneity, High chance of publication bias. Only trials using no treatment controls included elevating risk of Hawthorne effect. No conclusions can be drawn with regard to relative efficacy of Reminiscence Therapy for late life depression.

**KEY**

SMD = standard mean effect size. 95% CI = 95% Confidence Interval. Q = measure of heterogeneity. X<sup>2</sup> = Chi Square. FsI = Fail-safe N. FsI <40 indicates high chance of publication bias.

**Wilson et al., (2008)** *Psychotherapeutic treatments for older depressed people*

Analysis	Results			
	N	outcome	WMD	95% CI
Cognitive Behavioural Therapies Vs Control	153	HRDS	-9.85*	-11.97 to -7.73
Problem Solving Therapy Vs Control	39	GDS	-4.80	-8.32 to -1.28
Cognitive therapy Vs Behaviour therapy	78	HRDS	0.63	-4.29 to 3.04
Cognitive therapy Vs Psychodynamic therapy	57		-1.57	-5.59 to 2.44

**Main Effect**

CBT more effective than waiting list controls. No significant difference between psychodynamic therapy and CBT.

Sub-Group Analyses	N	WMD	95% CI
Bibliotherapy Vs Waiting list controls	86	-9.29*	-11.65 to -6.93
Cognitive therapy Vs Active Controls measured with HDRS	53	-5.69*	-11.04 to -0.35
Cognitive therapy Vs Active Controls measured with GDS	80	2.00	-5.31 to 1.32

**Sub-group analyses**

Bibliotherapy more effective than waiting list controls. CBT superior to active control when using the HRSD, but equivalent when using the GDS. Treatment approach did not predict dropout.

Dropout	Tr	Co	OR	95% CI	X <sup>2</sup>	df	p	Z	p
CBT vs Control	243	221	0.43	0.27 to 0.68	14.59	5	0.01	3.57	0.00035
CBT vs Active Control	68	52	1.19	0.55 to 2.59	1.26	2	0.53	0.45	0.65
CT vs BT	64	59	0.58	0.27 to 1.27	3.29	2	0.19	1.36	0.17
CogB vs Control	48	44	0.85	0.32 to 2.24	2.87	2	0.24	0.34	0.74
CBT vs PsyD therapy	63	54	1.01	0.43 to 2.35	5.10	1	0.02	0.02	0.98

**KEY**

\* Statistically significant. N = number of participants. HRSD = Hamilton Depression Rating Scale GDS = Geriatric Depression Scale. WMD = Weighted Mean Difference. 95 % CI = 95% Confidence Interval. Df = degrees of freedom. P = measure of significance. X<sup>2</sup> = Chi Square. OR = odds Ratio. Z = test for overall effect. PsyD = Psychodynamic. CogB = Cognitive bibliotherapy.

**Peng et al., (2009)** *Cognitive behavioural therapy and reminiscence techniques for the treatment of depression in the elderly: a systematic review*

Analysis	Results						
	N	SMD	95% CI	X <sup>2</sup>	df	I <sup>2</sup>	z
Cognitive Behavioural Therapy Vs Placebo/No intervention	NR	-1.34	-1.89 to -0.79	16.49	6 (p=0.01)	63.6%	4.8**
General Psychotherapy Vs Placebo/No intervention	NR	-1.00	-1.40 to -0.59	1.61	2 (p=0.45)	0%	4.34**
Reminiscence Vs Placebo/No intervention	NR	-0.64	-1.04 to -0.25	5.53	3 (p=0.14)	45.8%	3.2*
Cognitive Behavioural Therapy Vs Reminiscence	NR	-0.21	-0.61 to 0.20	1.83	3 (p=0.61)	0%	1 (p=0.32)

**Main Effect**

CBT, Reminiscence and 'General Psychotherapy' more effective than placebo/no intervention in decreasing depression scores. No significant difference between efficacy of CBT and Reminiscence Therapy.

**KEY**

\* = P<0.001 \*\* = P<0.00001. NR = Not reported. SMD = Standard mean effect size. 95 % CI = 95% Confidence Interval. X<sup>2</sup> = Chi Square. Df = degrees of freedom. I<sup>2</sup> the percentage of variation across studies that is due to heterogeneity rather than chance. OR = odds Ratio. Z = test for overall effect.

Dropout	OR	95% CI	X <sup>2</sup>	df	p	Z	p
Drug treatment with/without psychotherapy	1.03	0.55 to 1.94	5.69	4	0.22	0.11	0.92

**Cuijpers et al., (2006)**

*Psychological treatment of late-life depression: a meta-analysis of randomized controlled trials.*

Analysis	Results			
	d	95%CI	Q	I <sup>2</sup> (%)
All studies	0.72	0.69-0.85	101.3**	80.2
All studies (minustwo outliers)	0.72	0.57-0.87	17.28	1.6
<b>Inclusion criteria</b>				
MDD*	0.84	0.56-1.11	8.27	27.4
Other	0.67	0.49-0.85	7.24	0
<b>Format</b>				
Individual	0.73	0.51-0.95	7.42	19.2
Group	0.70	0.46-0.95	5.51	9.3
Bibliotherapy	0.73	0.35-1.11	4.32	7.5
<b>Recruitment</b>				
Community	0.77	0.60-0.95	9.70	0
Other	0.58	0.29-0.86	6.3	20.6
<b>Type of treatment</b>				
CBT	0.70	0.48-0.92	10.33	22.5
Other Treatments	0.74	0.53-0.94	6.88	0
<b>Control Group</b>				
Waiting List	0.72	0.51-0.92	12.97	22.9
Care-as-usual	0.75	0.49-1.01	1.45	0
Other	0.64	0.19-1.09	2.70	0

**Main Effect**

Psychological treatments have moderate to large effects on late-life depression.

**Sub-group analyses**

Equivalence of effect found between individual, group or bibliotherapy formats or between CBT and other types of psychological treatment. Severity of depression did not predict outcome. The effects were comparable between self-rated and clinician rated depression outcomes. No impact of control group on ES found.

**KEY**

\* With or without minor depression or dysthymia. \*\* Only Q statistic found to be significant.

**Pinquart et al., (2006)**

*Treatments for later-life depressive conditions: a meta-analytic comparison of pharmacotherapy and psychotherapy.*

Analysis	Number of treated subgroups	Results			
		d	95%CI	t	Q
<b>Clinician rated depression</b>					
CBT	26	-1.22	-1.42 to -1.03	-12.40*	35.95
Other	9	-0.75	-1.01 to -0.49	-5.67*	8.07
<b>Self-rated depression</b>					
CBT	40	-0.88	-1.05 to -0.71	-10.11*	81.80*
Other	12	-0.69	-0.95 to -0.42	-5.13*	17.31

**Main Effect**

Psychotherapy and pharmacotherapy did not show strong difference in effect sizes: Both moderately large.

**Sub-group analyses**

Greater clinician rated depression improvement seen in mild-moderate depression, for psychotherapy group as compared with drug therapy: not found on self-rated measures. ES for CBT as rated on clinician rated measures was greater than the effects of other forms of psychotherapy. Results for self-rated measures show similar trend but with significant heterogeneity.

**KEY**

\* p<0.001. d = effect size. t = t statistic. Q = measure of heterogeneity. Significant values of 'Q' indicate significant homogeneity of effect size. 95% CI = 95% Confidence Interval. Q = measure of heterogeneity. Sign. = Significance.

**Pinquart & Sorensen (2001)** *How effective are psychotherapeutic and other psychosocial interventions with older adults?*

Analysis		Results				
Intervention	N	g	95% CI	t	Homogeneity	
<b>Self-rated Depression</b>						
Cognitive Behavioural Therapy	436	0.64	0.50 to 0.78	9.3***	53.61**	
Psychodynamic Therapy	56	0.79	0.37 to 1.21	3.66***	0.98	
Reminiscence	386	0.44	0.30 to 0.58	6.17***	15.58	
<b>Clinician Rated Depression</b>						
Cognitive Behavioural Therapy	278	1.18	0.99 to 1.36	12.30***	35.23*	
Psychodynamic Therapy	39	1.68	1.10 to 2.27	5.62***	10.95***	
Reminiscence	21	0.66	-0.11 to 1.43	1.67	5.35*	

**Main Effect**

CBT and Psychodynamic approaches were associated with significantly higher effect sizes than reminiscence approaches. However, significant heterogeneity was found in most analyses. The main effect for Psychodynamic therapy (self-rated measures) was not subject to significant heterogeneity.

**Moderator analyses**

Individual interventions, Interventions with depressed, rather than non-depressed elders and use of clinician rated (HRSD) rather than self-rated (BDI/GDS) outcome measures were significantly associated with greater effect sizes. Therapists with advanced degree + gerontological experience associated with significantly higher effect sizes as compared with those with advanced degree / graduate / paraprofessionals. Neither type of control used nor study length, quality or setting were not found to moderate effect sizes.

Moderator Analyses												
		Participants		g		95% CI		t		Q		Sign.
		I	G	I	G	I	G	I	G	I	G	
<i>Individual (I) Vs Group (G) Conditions</i>												
Self-rated Depression	365	784	0.70	0.44	0.50 to 0.85	0.34 to 0.54	9.32***	8.71***	11.51	80.24***		✓
Clinician rated Depression	171	158	1.56	0.68	1.30-1.82	0.44 to 0.91	13.29***	5.93***	40.20***	14.21		✓
<i>Non-Depressed (ND) Vs Depressed (D)</i>												
Self-rated Depression	559	665	0.31	0.7	0.19 to 0.49	0.59 to 0.81	5.20***	12.63***	49.55*	38.24		✓
Clinician rated Depression	35	378	0.40	1.16	-0.90 to 0.86	1.00 to 1.32	1.68+	15.21***	-	51.60**		✓
<i>Community (C) Vs Nursing Home (NH)</i>												
Self-rated Depression	837	226	0.51	0.39	0.41 to 0.60	0.20 to 0.58	10.26***	4.30***	71.66*	12.47		
Clinician rated Depression	338	-	1.23	-	1.06 to 1.40	-	15.10***	-	47.13**	-		
<i>≤9 sessions (≤9) Vs &gt;9 sessions (&gt;9)</i>												
Self-rated Depression	520	571	0.40	0.59	0.28 to 0.52	0.47 to 0.71	6.52***	9.66***	34.52**	61.59**		
Clinician rated Depression	124	213	0.76	1.21	0.50 to 1.01	0.99 to 1.43	5.92***	11.88***	5.37	41.24***		
<i>Immediate posttest (Ipt) Vs Delayed posttest (Dpt)</i>												
Self-rated Depression	1040	184	0.55	0.34	0.46 to 0.64	0.13 to 0.54	12.55***	3.29***	85.86**	13.06		

Qualification	n	g	95% CI	t	Q	Sign. of difference
1. Graduate students/paraprofessionals	161	0.28	0.07 to 0.50	2.59*	10.77	
2. Advanced degree	316	0.40	0.24 to 0.56	5.04***	20.45	1,2 <3
3. Advanced degree + Gerontological experience	108	0.81	0.56 to 1.06	6.58***	10.95	
<b>Report Quality</b>						
1. Low	263	0.35	0.18 to 0.52	4.17***	32.59***	
2. Medium	680	0.56	0.45 to 0.68	10.13***	51.41	Not significant
3. High	241	0.56	0.37 to 0.74	6.00***	14.08	

**KEY**

\*\*\* p<0.001; \*\*p<0.01; \*p<0.05. g = mean effect size. t = t statistic 95% CI = 95% Confidence Interval. Q = measure of heterogeneity. Sign. = Significance.

**Cuijpers (1998)** *Psychological outreach programmes for the depressed elderly: a meta-analysis of effects and dropout*

Analysis	Results			
	d	95%CI	Z score	OrFslN
Treatment Vs Control	0.77	0.55-0.98	7.05 p<0.001	40
Pre-Post difference (n=473)	1.07	0.92-1.22	13.8 p<0.001	104
ES of studies with control compared with studies without control	1.14 vs 1.06 = non significant.			
ES of studies with formal diagnosis of Major depression compared with others	1.25 vs 0.9 = non significant.			
ES. CBT vs other therapies	0.2 = non significant			
ES. BT vs other therapies	0.05 = non significant.			
ES. Post-test to follow up (8 studies) 1, 3, 6 months.	-0.07, 0.02,0.03 non significant			

Regression analyses	$\beta$	SE
Intervention type, participant characteristics. Only one variable reached a significant level: CBT intervention.	0.35	0.16

Regression analyses of Drop out showed four significant predictors, together predicting 94% of the variance:	$\beta$	SE
Group	0.36	0.06
CBT	0.29	0.05
% Female	0.02	0.003
Number of sessions	0.04	0.01

**Main Effect**

Psychological treatment for depressed elders in the community is effective d=0.77.

**Further Analyses**

Regression analysis showed CBT to be a predictor of increased effect size. Format, N of sessions, depression severity, gender, mean age were non-significant predictors. CBT not significantly more effective in comparison with other approaches. Drop out significantly larger in: group interventions; CBT interventions; interventions with more sessions; conditions with a greater % women.

**KEY**

d = Effect Size. 95% CI = 95% Confidence Interval. OrFslN = Orwin's Fail safe N: number of studies with a zero-effect that should be found in order to reduce the effect size to 0.20.  $\beta$  = Regression Coefficient. SE = Standard Error. CBT = Cognitive Behaviour Therapy

**Bohlmeijer et al., (2003)** *Effects of reminiscence and life review on late-life depression: a meta-analysis*

Analysis	Results				
	N	d	95%CI	Q	SE%
Reminiscence and life review effect on depressive symptoms: All studies. (Z = 4.94 p<0.001 OrFslN = 74)	959	0.84	0.31-1.37	0.103.51***	12
All studies, outlier excluded	933	0.67	0.41-0.93	75.53***	39.5
For those participants with high depressive symptoms	391	1.23	0.92-1.53	9.59	70
For those participants without high depressive symptoms	542	0.37	0.12-0.62	25.71	60.3
High quality studies	240	0.92	0.28-1.56	14.25	25.5
Other studies	693	0.60	0.33-0.88	61.26***	46.7
Reminiscence	421	0.46	0.16-0.76	29.71**	56.2
Life-review	512	0.92	0.49-1.35	32.34***	27.7%
Group intervention	448	0.68	0.38-0.98	25.95	49.2
Individual intervention	485	0.64	0.11-1.17	49.58	20
Community residents	556	1.11	0.12-2.10	87.83***	21
Nursing/residential homes	325	0.38	0.05-0.71	7.149	63
Less than 72% women	516	0.75	0.21-1.28	59.193***	21
More than 72% women	417	0.58	0.31-0.84	18.085	66
Published studies	738	0.77	0.47-1.07	53.280***	39
Dissertations	195	0.20	0.17-0.58	9.262	64

**Main Effect**

Overall effect size of 0.84 indicated clinically significant effect of reminiscence and life review on depressive symptoms in elderly people although test for heterogeneity indicates significant variance attributable to the systematic effects of covariates.

**Sub-group analyses**

Larger effect in subjects with elevated depressive symptom (d1.23) as compared to other subjects (d0.37). All other sub-group comparisons overlap at 95% CI: rem vs life review; high vs low quality; group vs individual; community vs non-community; studies >72% women vs <72% women; published vs unpublished.

**KEY**

\*\*\* p<0.001; \*\*p<0.01; \*p<0.05. d = effect size. CI = 95% Confidence Interval. Q = Cochran's Q measure of heterogeneity. SE = percentage of the variance accounted for by random sample error. OrFslN = Orwin's Failsafe N: number of studies with a zero-effect that should be found in order to reduce the effect size to 0.20.

Engels & Vermey (1997) *Efficacy of nonmedical treatments of depression in elders: A quantitative analysis*

Analysis	Results				
	Number of therapies	Number of Individuals	d	Fisher Z	Variability of ES (p)
Therapies compared with real controls	20	405	0.52	0.26	0
Therapies compared with artificial controls	8	66	1.06	0.51	0.07
Difference between real and artificial controls	Z= 2.65 p = 0.00				
<b>Types of therapy (using real controls)</b>					
Cognitive	7	128	0.78	0.38	0.22
Behaviour	3	57	0.85	0.41	0.09
Cognitive/Behaviour	3	34	0.12	0.06	0.08
Psychodynamic	1	32	0.61	0.3	na
Rest	6	162	0.28	0.14	0.01
<b>Mode <sup>a</sup></b>					
Therapy mode: individual.	18 (12 <sup>*</sup> )	348	0.76	0.37	0.12
Therapy mode: group.	9 (7 <sup>*</sup> )	111	0.16	0.08	0.00
Therapy mode: individual & group	1 <sup>*</sup>	12	0.71	0.35	na
Mean effect size of individual Vs Group (including artificial controls)/(excluding artificial controls)	(Z = 3.74 p = 0.00 N =27) / (Z = 4.41 p = 0.00 N =19)				
<b>Diagnosis <sup>a</sup></b>					
Major Depression	10 (5 <sup>*</sup> )		0.86	0.42	0.14
Depression	10 <sup>*</sup>		0.68	0.33	0.21
Multiple Complaints	8 (5 <sup>*</sup> )		0.14	0.07	0.00
<b>Severity of Depression <sup>a</sup></b>					
Mild/Moderate Depression	9 (7 <sup>*</sup> )	103	0.72	0.35	0.01
Severe Depression	12 (8 <sup>*</sup> )	258	0.68	0.33	0.32
Mild/Moderate Depression Vs Severe depression (All studies/ Controlled studies)	(Z=0.83, p=0.20, N=21) / Z=0.44, p= 0.33, N = 15)				
<b>Type of control <sup>a</sup> controls (n)</b>					
No treatment	10	144	0.64	0.32	0.00
Placebo	6	117	0.28	0.31	0.31
No treatment Vs Control	Z= 2.08 p = 0.02, N=16				
<b>Age <sup>a</sup> The mean age in controlled/uncontrolled studies differed significantly.</b>					
Including artificial controls in analysis	(r =-0.03, p =0.41, N=28)				
Excluding artificial controls	(r =-0.33, p =0.01, N=20)				
<b>Type of depression measure<sup>§</sup></b>					
HRSD	14 (9 <sup>*</sup> )		1.10	0.52	0.02
BDI-21	16(11 <sup>*</sup> )		0.57	0.28	0.00
BDI-13	11 (6 <sup>*</sup> )		0.62	0.31	0.39
GDS	7(6 <sup>*</sup> )		0.68	0.33	0.00
Rest	7 <sup>*</sup>		0.65	0.32	0.15
HRSD Vs BDI-21	Z= 3.30, p=0.00				
HRSD Vs BDI-13	Z=2.14, p=0.02				
HRSD Vs GDS	Z=1.56, p=0.05				

**Main Effect**

This meta-analysis included non-medical treatments for late-life depression such as anger expression, music therapy and physical training. The overall effect size is therefore not reported here. In addition, comparisons were made using real and artificial control conditions. However, artificial controls were shown to result in significantly higher effect sizes. Results therefore need to be interpreted with caution. Where comparisons have included artificial controls the number of studies for which real controls were used is indicated. Overall effect sizes: Cognitive therapy d= 0.78; Behaviour therapy, d = 0.85; Cognitive and Behaviour Therapy, d =0.12; Psychodynamic therapy, d = 0.61

**Sub-group analyses**

Individual rather than group treatment and use of clinician rated outcome (HRSD) rather than self-rated (BDI/GDS) were significantly associated with greater effect sizes. Comparisons with placebo yielded significantly lower effect sizes than no-treatment or waiting list controls. Severity, age, gender, length of treatment yielded no evidence of moderating effect. Those with multiple complaints were found to respond less well to treatment. Age: The mean age in controlled/uncontrolled studies differed significantly. Including artificial controls in analysis yielded non significant correlation (r =-0.03, p =0.41, N=28). Excluding artificial controls it was significant (r =-0.33, p =0.01, N=20). No firm conclusions can be drawn about age effects.

**KEY**

<sup>a</sup> = Analyses include non-psychological treatments. D= Mean effect sizes. ES = Effect Size. \* = number with real controls. HRSD = Hamilton Rating Scale for Depression. BDI= Becks Depression Inventory. GDS = Geriatric Depression Scale

**Gorey & Cryns (1991)** *Group work as interventive modality with the older depressed client: A meta-analytic review*

Analysis	Results					
	Mean ES	SD	d	Cohen's U <sub>s</sub> (%)	r-index	r <sup>2</sup>
Group (Pre-Post Comparison)	0.98	0.51	0.92	82.22	0.42	0.17
Group Vs No treatment	0.62	0.51	0.68	75.16	0.32	
Group Vs Placebo	0.09	0.53	0.09	53.6	0.04	
Group Vs Drug Intervention	-0.63	0.69	-0.67	-74.84		
(Group Vs No treatment r-index)/(Group Vs Placebo r-index)						0.32/0.42 = 0.76
(Group Vs Placebo r-index)/(Group Vs No treatment r-index)						0.04/0.32 = 0.13
Moderator Effects						
Overall heterogeneity	Chi-Square (18) = 52.95 p<0.001					
Publication date	Inversely related to ES (r = 0.52, p = 0.024)					
Low to high study quality	ES attenuated by 2/3. (F 3.18) = 9.90, p = 0.001)					
Drop out 0-15% / 16%-50%	d = 1.26 / 0.31					
Studies where >25% of participants lived alone / <25%	d = 1.42 / 0.84		(F(1,10) = 5.50, p = 0.41)			
Depression narratively defined as mild/moderate/severe	d = 0.14/0.94/1.37		(F(2,18) = 7.07, p = 0.05)			
Group size: <6/ 6-14	d = 1.38/0.81		(F(1,16) = 6.49, p = 0.22)			
Publication bias:	Failsafe N at p=0.05 was calculated at 90 thus falling short of Rosenthal's criterion: (Included studies) + 10 = 105. Although one way ANOVA on ES by book/journal Vs dissertation reported as non-significant (F1.17 = 0.04)					

**Main Effect**

Analyses of impact of non-specific factors undertaken using a comparison of r-index and r<sup>2</sup> (coefficient of determination). Conclusion: Group intervention only accounts for 17% of variance in affective state on pre-post test comparison. Comparing groups with no treatment controls and groups with placebo controls it was inferred that 76% of change is accounted for by interventive factors (specific and non-specific). Comparing groups with placebo controls with groups with no treatment it is inferred that 13% of improvement in depressive symptoms is due to intervention specific effects leaving 87% of overall improvement due to non-specific factors.

**Further analyses**

Increased severity of depression, living alone, waiting list control (rather than active control), poorer study quality and smaller groups reported to be significant predictors of outcome in group therapy. Type, duration and setting of therapy, and age and gender of participants found to be non-significant predictors of outcome (methods not reported).

**KEY**

ES = Effect Size. SD = Standard deviation. d = 'unbiased' ES. Cohen's U<sub>s</sub>: % of participants scoring below the mean pre-intervention score. R index: converted to % indicates % improvement of depressive symptoms. R<sup>2</sup> = coefficient of determination: the % of variance accounted for by intervention. F = explained variance/unexplained variance.

**Scogin & McElreath (1994)** *Efficacy of psychosocial treatments for geriatric depression: a quantitative review*

Analysis	Results			
	ES	Z score	WMES	FsN
Overall effect size: treatment Vs no treatment/placebo	0.78	(Z = 3.74, p < .05)	1.22	2658
Major Depression (four studies)	0.76	(Z = 1.70, p < .05)	0.87	22
Clinical Depression and Sub-clinical Depression group (18 remaining studies)	0.79	(Z = 3.35, p < .05)	1.35	1326
CBT (including individual, group, and self-administered)	0.85	(Z = 2.08, p < .05)	0.89	52
Reminiscence Therapy	1.05	(Z = 2.97, p < .05)	1.92	201
Self-report outcome measures (treatment versus no-treatment or placebo)	0.69	(Z = 3.31, p < .05)	1.10	2,119
Other-rated measures (treatment versus no-treatment or placebo)	1.15	(Z = 3.64, p < .05)	1.33	480
Group treatment (n = 13), .	0.74	(Z = 2.56, p < .05), .	1.58	337
Individual treatment (n = 7)	0.77	(Z = 2.04, p < .05)	0.85	68
Cognitive Approaches Vs Other Approaches (n = 7 including IPT and BT)	0.41	(Z = 1.09, p > .05).	0.30	
Behavioural approaches Vs Other Approaches (n = 6 including CT, IPT, Supportive)	0.00	(p > .05).	-0.06	
Treatment Vs No treatment. Correlation between treatment quality and effect size (n=14)	0.19 (n = 14),			
Association of effect size to number of treatment sessions across treatment versus no-treatment comparisons.	-0.20 (n = 12)			

**Main Effect**

Psychosocial interventions more effective than no-treatment or placebo in decreasing depressive symptoms in older adults. Effect size: 0.78

**Further Analyses**

No correlation between effect sizes and type of therapy, severity of depression, therapy modality, duration of therapy and study quality.

**KEY**

ES = Effect Size. WMES = Weighted Mean Effect Size. FsN = Fail-safe N



**Krishna et al., (2011).** *Is group psychotherapy effective in older adults with depression? A systematic review*

Analysis	Results				
	MD	Z	p	95%CI	t <sup>2</sup>
Group Psychotherapy treatment versus (any) control.	3.92	3.41	0.001	-6.18 to -1.67	14.17
Group psychotherapeutic treatment versus waiting list control	6.29	4.63	0.0001	-8.95 to -3.62	6.68
Group psychotherapeutic treatment versus other therapeutic treatments	2.74	2.21	0.03	-5.16 to -0.31	10.61
Group Psychotherapy versus active control: completion to follow-up	-0.2	0.18	0.86	-2.06 to 1.67	0
Pooled					
Main measures of effect of intervention Vs waiting list	MD	SD	ES <sup>+</sup>	95%CI	p
Combined measures (6)	6.26	6.91	-0.86	-1.50 to 0.21	p<0.01
Geriatric depression scale (3)	4.76	6.18	-0.75	-1.39 to -0.11	p<0.01
Beck depression inventory (2)	5.5	6.95	-0.77	-1.52 to -0.02	p<0.01
<b>Main measures of effect of intervention Vs active control</b>					
Combined measures (11)	3.1	7.49	-0.36	-1.09 to 0.37	p<0.05
Geriatric depression scale (4)	1.18	5.94	-0.10	-0.86 to 0.37	
Hamilton depression rating scale (3)	7.84	7.52	-0.95	-1.75 to -0.14	p<0.01
Beck depression inventory (4)	1.47	9.02	-0.19	-0.82 to 0.45	

**Main Effect**

Group psychotherapy is an effective intervention for late-life depression compared with waiting list controls but with a very modest effect size. Group intervention versus active interventions did not reach statistical significance indicating no effect of the intervention versus all variations in the active control conditions.

**Further analyses**

Waiting list control had significantly fewer losses to follow up than intervention groups. HDRS outcome measure found significant difference favouring the group therapies compared to active controls but this was not found with BDI and GDS outcome measures.

**KEY**

MD = Mean effect size. SD = Standard deviation. ES = Effect Size. ES<sup>+</sup> = Bias corrected ES. 95 % CI = 95% Confidence Interval. Z = z score. t<sup>2</sup> = t<sup>2</sup> distribution.

**Samad et al., (2011)** *The effectiveness of behavioural therapy for the treatment of depression in older adults: a meta-analysis.*

Analysis	Results						
	WMD	95%CI	p	X <sup>2</sup>	df	p	I <sup>2</sup> +
Self-rated effectiveness of behaviour therapy Vs WL control at treatment completion	-0.52	-1.35 to 0.30	0.21	8.94	2	0.01	78%
Clinician rated effectiveness of behaviour therapy Vs waiting list control at treatment completion	-5.86	-7.71 to -3.66	0.001	0.11	2	0.94	0%
Behaviour therapy Vs cognitive therapy							
Self-rated effectiveness of behaviour therapy V cognitive therapy at follow up (1-3 months)	0.23	-0.24 to 0.70	0.33	5.24	3	0.15	43%
Self-rated effectiveness of behaviour therapy V cognitive therapy at follow up (1-3 months): Only studies with formal diagnosis of depression.	0.02	-0.44 to 0.48	0.93				
Clinician rated effectiveness of behaviour therapy Vs cognitive therapy at treatment completion	-0.05	-2.10 to 2.00	0.96	3.45	3	0.33	21%
Clinician rated effectiveness of behaviour therapy Vs cognitive therapy at treatment completion: Only studies with formal diagnosis of depression.	-1.25	-4.30 to 1.79	0.42				
Self-rated effectiveness of behavioural therapy Vs brief psycho-dynamic therapy at post-treatment (or 3 month follow-up).	-0.37	-0.84 to 0.11	0.13	0.00	1	0.96	0%
Clinician rated effectiveness of behavioural therapy Vs brief psycho-dynamic therapy at post-treatment (or 3 month follow-up).	-1.56	-4.64 to 1.52	0.32	0.24	1	0.62	0%

**Main Effect**

Behavioural therapy for older people significantly more effective than WL control when measured by clinician-rated depression (HDRS) but not significantly different when measured by patient self-report (BDI & GDS)

**Further analyses**

Excluding studies without a formal diagnosis of depression at baseline reduced the effectiveness of cognitive therapy compared to behavioural therapy (In self-reported depression). Analysis of drop-out: pooled dropout ratio CT Vs BT (OR = 2.04 95% CI 0.87 to 4.78, p=0.10) and for BT Vs BPT (OR = 1.50 (95% CI 0.32–6.96, p=0.61) both non-significant.

**KEY**

WMD = Weighted mean effect size. CI = 95% Confidence Interval. X<sup>2</sup> = Chi square. df = degrees of freedom. I<sup>2</sup> = the percentage of variation across studies that is due to heterogeneity rather than chance.

## Appendix 5: PRISMA Guidelines (Systematic Review).



## Guidelines and Guidance

# Preferred Reporting Items for Systematic Reviews and Meta-Analyses: The PRISMA Statement

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## Introduction

Systematic reviews and meta-analyses have become increasingly important in health care. Clinicians read them to keep up to date with their field [1,2], and they are often used as a starting point for developing clinical practice guidelines. Granting agencies may require a systematic review to ensure there is justification for further research [3], and some health care journals are moving in this direction [4]. As with all research, the value of a systematic review depends on what was done, what was found, and the clarity of reporting. As with other publications, the reporting quality of systematic reviews varies, limiting readers' ability to assess the strengths and weaknesses of those reviews.

Several early studies evaluated the quality of review reports. In 1987, Mulrow examined 50 review articles published in four leading medical journals in 1985 and 1986 and found that none met all eight explicit scientific criteria, such as a quality assessment of included studies [5]. In 1987, Sacks and colleagues [6] evaluated the adequacy of reporting of 83 meta-analyses on 23 characteristics in six domains. Reporting was generally poor; between one and 14 characteristics were adequately reported (mean = 7.7; standard deviation = 2.7). A 1996 update of this study found little improvement [7].

In 1996, to address the suboptimal reporting of meta-analyses, an international group developed a guidance called the QUOROM Statement (*QU*ality *O*f *R*eporting *O*f *M*eta-analyses), which focused on the reporting of meta-analyses of randomized controlled trials [8]. In this article, we summarize a revision of these guidelines, renamed PRISMA (Preferred Reporting Items for Systematic reviews and Meta-Analyses), which have been updated to address several conceptual and practical advances in the science of systematic reviews (Box 1).

## Terminology

The terminology used to describe a systematic review and meta-analysis has evolved over time. One reason for changing the name from QUOROM to PRISMA was the desire to encompass both systematic reviews and meta-analyses. We have adopted the definitions used by the Cochrane Collaboration [9]. A systematic review is a review of a clearly formulated question that uses systematic and explicit methods to identify, select, and critically appraise relevant research, and to collect and analyze data from the studies that are included in the review. Statistical methods (meta-analysis) may or may not be used to analyze and summarize the results of the included studies. Meta-analysis refers to the use of statistical techniques in a systematic review to integrate the results of included studies.

## Developing the PRISMA Statement

A three-day meeting was held in Ottawa, Canada, in June 2005 with 29 participants, including review authors, methodologists,

clinicians, medical editors, and a consumer. The objective of the Ottawa meeting was to revise and expand the QUOROM checklist and flow diagram, as needed.

The executive committee completed the following tasks, prior to the meeting: a systematic review of studies examining the quality of reporting of systematic reviews, and a comprehensive literature search to identify methodological and other articles that might inform the meeting, especially in relation to modifying checklist items. An international survey of review authors, consumers, and groups commissioning or using systematic reviews and meta-analyses was completed, including the International Network of Agencies for Health Technology Assessment (INAHTA) and the Guidelines International Network (GIN). The survey aimed to ascertain views of QUOROM, including the merits of the existing checklist items. The results of these activities were presented during the meeting and are summarized on the PRISMA Web site (<http://www.prisma-statement.org/>).

Only items deemed essential were retained or added to the checklist. Some additional items are nevertheless desirable, and review authors should include these, if relevant [10]. For example, it is useful to indicate whether the systematic review is an update [11] of a previous review, and to describe any changes in procedures from those described in the original protocol.

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**Competing Interests:** The authors have declared that no competing interests exist.

**Abbreviations:** PRISMA, Preferred Reporting Items for Systematic reviews and Meta-Analyses; QUOROM, *QU*ality *O*f *R*eporting *O*f *M*eta-analyses.

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## Box 1: Conceptual Issues in the Evolution from QUOROM to PRISMA

**Completing a Systematic Review Is an Iterative Process** The conduct of a systematic review depends heavily on the scope and quality of included studies: thus systematic reviewers may need to modify their original review protocol during its conduct. Any systematic review reporting guideline should recommend that such changes can be reported and explained without suggesting that they are inappropriate. The PRISMA Statement (Items 5, 11, 16, and 23) acknowledges this iterative process. Aside from Cochrane reviews, all of which should have a protocol, only about 10% of systematic reviewers report working from a protocol [22]. Without a protocol that is publicly accessible, it is difficult to judge between appropriate and inappropriate modifications.

**Conduct and Reporting Research Are Distinct Concepts** This distinction is, however, less straightforward for systematic reviews than for assessments of the reporting of an individual study, because the reporting and conduct of systematic reviews are, by nature, closely intertwined. For example, the failure of a systematic review to report the assessment of the risk of bias in included studies may be seen as a marker of poor conduct, given the importance of this activity in the systematic review process [37].

**Study-Level Versus Outcome-Level Assessment of Risk of Bias** For studies included in a systematic review, a thorough assessment of the risk of bias requires both a “study-level” assessment (e.g., adequacy of allocation concealment) and, for some features, a newer approach called “outcome-level” assessment. An outcome-level assessment involves evaluating the reliability and validity of the data for each important outcome by determining the methods used to assess them in each individual study [38]. The quality of evidence may differ across outcomes, even within a study, such as between a primary efficacy outcome, which is likely to be very carefully and systematically measured, and the assessment of serious harms [39], which may rely on spontaneous reports by investigators. This information should be reported to allow an explicit assessment of the extent to which an estimate of effect is correct [38].

**Importance of Reporting Biases** Different types of reporting biases may hamper the conduct and interpretation of systematic reviews. Selective reporting of complete studies (e.g., publication bias) [28] as well as the more recently empirically demonstrated “outcome reporting bias” within individual studies [40,41] should be considered by authors when conducting a systematic review and reporting its results. Though the implications of these biases on the conduct and reporting of systematic reviews themselves are unclear, some previous research has identified that selective outcome reporting may occur also in the context of systematic reviews [42].

Shortly after the meeting a draft of the PRISMA checklist was circulated to the group, including those invited to the meeting but unable to attend. A disposition file was created containing comments and revisions from each respondent, and the checklist was subsequently revised 11 times. The group approved the checklist, flow diagram, and this summary paper.

Although no direct evidence was found to support retaining or adding some items, evidence from other domains was believed to be relevant. For example, Item 5 asks authors to provide registration information about the systematic review, including a registration number, if available. Although systematic review registration is not yet widely available [12,13], the participating journals of the International Committee of Medical Journal Editors (ICMJE) [14] now require all clinical trials to be registered in an effort to increase transparency and accountability [15]. Those aspects are also likely to benefit systematic reviewers, possibly reducing the risk of an excessive number of reviews addressing the same question [16,17] and providing greater transparency when updating systematic reviews.

## The PRISMA Statement

The PRISMA Statement consists of a 27-item checklist (Table 1; see also Text S1 for a downloadable Word template for researchers to re-use) and a four-phase flow diagram (Figure 1; see also Figure S1 for a downloadable Word template for researchers to re-use). The aim of the PRISMA Statement is to help authors improve the reporting of systematic reviews and meta-analyses. We have focused on randomized trials, but PRISMA can also be used as a basis for reporting systematic reviews of other types of research, particularly evaluations of interventions. PRISMA may also be useful for critical appraisal of published systematic reviews. However, the PRISMA checklist is not a quality assessment instrument to gauge the quality of a systematic review.

## From QUOROM to PRISMA

The new PRISMA checklist differs in several respects from the QUOROM checklist, and the substantive specific changes are highlighted in Table 2. Generally, the PRISMA checklist “decouples” several items present in the QUOROM checklist and, where applicable, several checklist items are linked to improve consistency across the systematic review report.

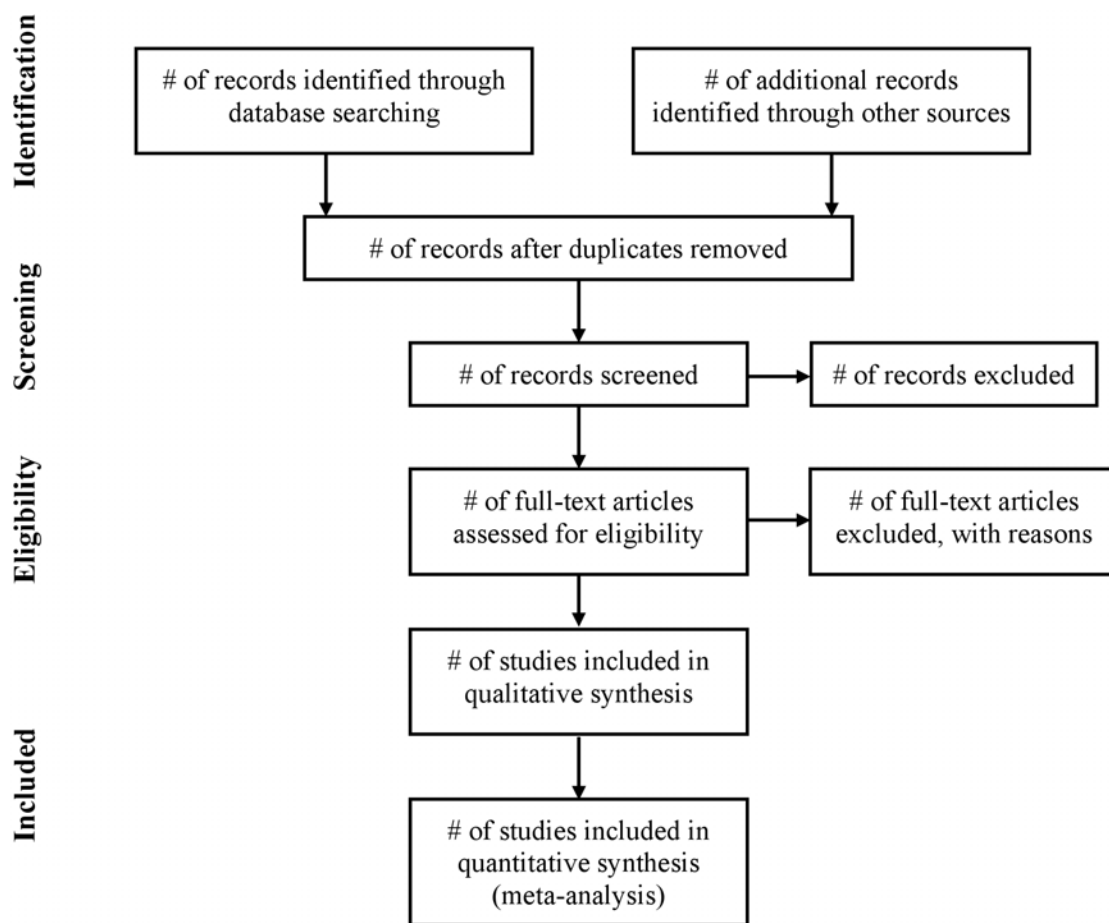
The flow diagram has also been modified. Before including studies and providing reasons for excluding others, the review team must first search the literature. This search results in records. Once these records have been screened and eligibility criteria applied, a smaller number of articles will remain. The number of included articles might be smaller (or larger) than the number of studies, because articles may report on multiple studies and results from a particular study may be published in several articles. To capture this information, the PRISMA flow diagram now requests information on these phases of the review process.

## Endorsement

The PRISMA Statement should replace the QUOROM Statement for those journals that have endorsed QUOROM. We hope that other journals will support PRISMA; they can do so by registering on the PRISMA Web site. To underscore to authors, and others, the importance of transparent reporting of systematic reviews, we encourage supporting journals to reference the PRISMA Statement and include the PRISMA Web address in their Instructions to Authors. We also invite editorial organizations to consider endorsing PRISMA and encourage authors to adhere to its principles.

## The PRISMA Explanation and Elaboration Paper

In addition to the PRISMA Statement, a supporting Explanation and Elaboration document has been produced [18] following the style used for other reporting guidelines [19–21]. The process



**Figure 1. Flow of information through the different phases of a systematic review.**  
doi:10.1371/journal.pmed.1000097.g001

of completing this document included developing a large database of exemplars to highlight how best to report each checklist item, and identifying a comprehensive evidence base to support the inclusion of each checklist item. The Explanation and Elaboration document was completed after several face to face meetings and numerous iterations among several meeting participants, after which it was shared with the whole group for additional revisions and final approval. Finally, the group formed a dissemination subcommittee to help disseminate and implement PRISMA.

## Discussion

The quality of reporting of systematic reviews is still not optimal [22–27]. In a recent review of 300 systematic reviews, few authors reported assessing possible publication bias [22], even though there is overwhelming evidence both for its existence [28] and its impact on the results of systematic reviews [29]. Even when the possibility of publication bias is assessed, there is no guarantee that systematic reviewers have assessed or interpreted it appropriately [30]. Although the absence of reporting such an assessment does not necessarily indicate that it was not done, reporting an assessment of possible publication bias is likely to be a marker of the thoroughness of the conduct of the systematic review.

Several approaches have been developed to conduct systematic reviews on a broader array of questions. For example, systematic

reviews are now conducted to investigate cost-effectiveness [31], diagnostic [32] or prognostic questions [33], genetic associations [34], and policy making [35]. The general concepts and topics covered by PRISMA are all relevant to any systematic review, not just those whose objective is to summarize the benefits and harms of a health care intervention. However, some modifications of the checklist items or flow diagram will be necessary in particular circumstances. For example, assessing the risk of bias is a key concept, but the items used to assess this in a diagnostic review are likely to focus on issues such as the spectrum of patients and the verification of disease status, which differ from reviews of interventions. The flow diagram will also need adjustments when reporting individual patient data meta-analysis [36].

We have developed an explanatory document [18] to increase the usefulness of PRISMA. For each checklist item, this document contains an example of good reporting, a rationale for its inclusion, and supporting evidence, including references, whenever possible. We believe this document will also serve as a useful resource for those teaching systematic review methodology. We encourage journals to include reference to the explanatory document in their Instructions to Authors.

Like any evidence-based endeavor, PRISMA is a living document. To this end we invite readers to comment on the revised version, particularly the new checklist and flow diagram, through the PRISMA Web site. We will use such information to inform PRISMA's continued development.

**Table 1.** Checklist of items to include when reporting a systematic review or meta-analysis.

Section/Topic	#	Checklist Item	Reported on Page #
<b>TITLE</b>			
Title	1	Identify the report as a systematic review, meta-analysis, or both.	
<b>ABSTRACT</b>			
Structured summary	2	Provide a structured summary including, as applicable: background; objectives; data sources; study eligibility criteria, participants, and interventions; study appraisal and synthesis methods; results; limitations; conclusions and implications of key findings; systematic review registration number.	
<b>INTRODUCTION</b>			
Rationale	3	Describe the rationale for the review in the context of what is already known.	
Objectives	4	Provide an explicit statement of questions being addressed with reference to participants, interventions, comparisons, outcomes, and study design (PICOS).	
<b>METHODS</b>			
Protocol and registration	5	Indicate if a review protocol exists, if and where it can be accessed (e.g., Web address), and, if available, provide registration information including registration number.	
Eligibility criteria	6	Specify study characteristics (e.g., PICOS, length of follow-up) and report characteristics (e.g., years considered, language, publication status) used as criteria for eligibility, giving rationale.	
Information sources	7	Describe all information sources (e.g., databases with dates of coverage, contact with study authors to identify additional studies) in the search and date last searched.	
Search	8	Present full electronic search strategy for at least one database, including any limits used, such that it could be repeated.	
Study selection	9	State the process for selecting studies (i.e., screening, eligibility, included in systematic review, and, if applicable, included in the meta-analysis).	
Data collection process	10	Describe method of data extraction from reports (e.g., piloted forms, independently, in duplicate) and any processes for obtaining and confirming data from investigators.	
Data items	11	List and define all variables for which data were sought (e.g., PICOS, funding sources) and any assumptions and simplifications made.	
Risk of bias in individual studies	12	Describe methods used for assessing risk of bias of individual studies (including specification of whether this was done at the study or outcome level), and how this information is to be used in any data synthesis.	
Summary measures	13	State the principal summary measures (e.g., risk ratio, difference in means).	
Synthesis of results	14	Describe the methods of handling data and combining results of studies, if done, including measures of consistency (e.g., $I^2$ ) for each meta-analysis.	
Risk of bias across studies	15	Specify any assessment of risk of bias that may affect the cumulative evidence (e.g., publication bias, selective reporting within studies).	
Additional analyses	16	Describe methods of additional analyses (e.g., sensitivity or subgroup analyses, meta-regression), if done, indicating which were pre-specified.	
<b>RESULTS</b>			
Study selection	17	Give numbers of studies screened, assessed for eligibility, and included in the review, with reasons for exclusions at each stage, ideally with a flow diagram.	
Study characteristics	18	For each study, present characteristics for which data were extracted (e.g., study size, PICOS, follow-up period) and provide the citations.	
Risk of bias within studies	19	Present data on risk of bias of each study and, if available, any outcome-level assessment (see Item 12).	
Results of individual studies	20	For all outcomes considered (benefits or harms), present, for each study: (a) simple summary data for each intervention group and (b) effect estimates and confidence intervals, ideally with a forest plot.	
Synthesis of results	21	Present results of each meta-analysis done, including confidence intervals and measures of consistency.	
Risk of bias across studies	22	Present results of any assessment of risk of bias across studies (see Item 15).	
Additional analysis	23	Give results of additional analyses, if done (e.g., sensitivity or subgroup analyses, meta-regression [see Item 16]).	
<b>DISCUSSION</b>			
Summary of evidence	24	Summarize the main findings including the strength of evidence for each main outcome; consider their relevance to key groups (e.g., health care providers, users, and policy makers).	
Limitations	25	Discuss limitations at study and outcome level (e.g., risk of bias), and at review level (e.g., incomplete retrieval of identified research, reporting bias).	
Conclusions	26	Provide a general interpretation of the results in the context of other evidence, and implications for future research.	
<b>FUNDING</b>			
Funding	27	Describe sources of funding for the systematic review and other support (e.g., supply of data); role of funders for the systematic review.	

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**Table 2.** Substantive specific changes between the QUOROM checklist and the PRISMA checklist (a tick indicates the presence of the topic in QUOROM or PRISMA).

Section/Topic	Item	QUOROM	PRISMA	Comment
Abstract		✓	✓	QUOROM and PRISMA ask authors to report an abstract. However, PRISMA is not specific about format.
Introduction	Objective		✓	This new item (4) addresses the explicit question the review addresses using the PICO reporting system (which describes the participants, interventions, comparisons, and outcome(s) of the systematic review), together with the specification of the type of study design (PICOS); the item is linked to Items 6, 11, and 18 of the checklist.
Methods	Protocol		✓	This new item (5) asks authors to report whether the review has a protocol and if so how it can be accessed.
Methods	Search	✓	✓	Although reporting the search is present in both QUOROM and PRISMA checklists, PRISMA asks authors to provide a full description of at least one electronic search strategy (Item 8). Without such information it is impossible to repeat the authors' search.
Methods	Assessment of risk of bias in included studies	✓	✓	Renamed from "quality assessment" in QUOROM. This item (12) is linked with reporting this information in the results (Item 19). The new concept of "outcome-level" assessment has been introduced.
Methods	Assessment of risk of bias across studies		✓	This new item (15) asks authors to describe any assessments of risk of bias in the review, such as selective reporting within the included studies. This item is linked with reporting this information in the results (Item 22).
Discussion		✓	✓	Although both QUOROM and PRISMA checklists address the discussion section, PRISMA devotes three items (24–26) to the discussion. In PRISMA the main types of limitations are explicitly stated and their discussion required.
Funding			✓	This new item (27) asks authors to provide information on any sources of funding for the systematic review.

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## Supporting Information

### Figure S1 Flow of information through the different phases of a systematic review (downloadable template document for researchers to re-use).

Found at: doi:10.1371/journal.pmed.1000097.s001 (0.08 MB DOC)

### Text S1 Checklist of items to include when reporting a systematic review or meta-analysis (downloadable template document for researchers to re-use).

Found at: doi:10.1371/journal.pmed.1000097.s002 (0.04 MB DOC)

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## Author Contributions

ICMJE criteria for authorship read and met: DM ALJT DGA. Wrote the first draft of the paper: DM AL DGA. Contributed to the writing of the

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## Appendix 6: Rating according to PRISMA criteria (Systematic Review).

Note: Text highlighted in red indicates PRISMA criteria which were assessed as being not met, or only partially met. Page numbers are given to facilitate cross-referencing original paper, with notes explaining rationale for scoring.

# PRISMA CHECKLIST

## Gorey & Cryns (1991)

			Page	Notes	Score
<b>Title</b>					
1	Title	Identify the report as a systematic review, meta-analysis, or both.	137	Yes	2
<b>Abstract</b>					
2	Structured summary	Provide a structured summary including, as applicable: background; objectives; data sources; study eligibility criteria, participants, and interventions; study appraisal and synthesis methods; results; limitations; conclusions and implications of key findings; systematic review registration number.	137	Partial	1
<b>Introduction</b>					
3	Rationale	Describe the rationale for the review in the context of what is already known.	137-139	Summary of what is known not detailed: partial	1
4	Objectives	Provide an explicit statement of questions being addressed with reference to participants, interventions, comparisons, outcomes, and study design (PICOS).	139	Outcomes and study design not included: partial	1
<b>Methods</b>					
5	Protocol and registration	Indicate if a review protocol exists, if and where it can be accessed (e.g., Web address), and, if available, provide registration information including registration number.	140	No	0
6	Eligibility criteria	Specify study characteristics (e.g., PICOS, length of follow-up) and report characteristics (e.g., years considered, language, publication status) used as criteria for eligibility, giving rationale.	140	No: explicit eligibility criteria not defined.	0
7	Information sources	Describe all information sources (e.g., databases with dates of coverage, contact with study authors to identify additional studies) in the search and date last searched.	140	Partial: dates of searches contact with study authors not included	1
8	Search	Present full electronic search strategy for at least one database, including any limits used, such that it could be repeated.	139	No	0
9	Study selection	State the process for selecting studies (i.e., screening, eligibility, included in systematic review, and, if applicable, included in the meta-analysis).	139-140	No	0
10	Data collection process	Describe method of data extraction from reports (e.g., piloted forms, independently, in duplicate) and any processes for obtaining and confirming data from investigators.	140	No	0
11	Data items	List and define all variables for which data were sought (e.g., PICOS, funding sources) and any assumptions and simplifications made.	140	No	0
12	Risk of bias in individual studies	Describe methods used for assessing risk of bias of individual studies (including specification of whether this was done at the study or outcome level), and how this information is to be used in any data synthesis.	140	Partial: Poor quality of trials noted, implicating but not outlining risks of bias. Fail-safe Ns described.	1
13	Summary measures	State the principal summary measures (e.g., risk ratio, difference in means).	140	Partial: Cohens d: Effect sizes.	1
14	Synthesis of results	Describe the methods of handling data and combining results of studies, if done, including measures of consistency (e.g., I <sup>2</sup> ) for each meta-analysis.		Homogeneity analysis undertaken, description condensed; replication difficult: Partial.	1
15	Risk of bias across studies	Specify any assessment of risk of bias that may affect the cumulative evidence (e.g., publication bias, selective reporting within studies).	141	No	0
16	Additional analyses	Describe methods of additional analyses (e.g., sensitivity or subgroup analyses, meta-regression), if done, indicating which were pre-specified	141	Sub-group analysis described, but not replicable: Partial	1



PRISMA CHECKLIST			Gorey & Cryns (1991)		
			Page	Notes	Score
<b>Results</b>					
17	Study selection	Give numbers of studies screened, assessed for eligibility, and included in the review, with reasons for exclusions at each stage, ideally with a flow diagram.	140	Reasons for exclusion not well described, and no flow chart: Partial	1
18	Study characteristics	For each study, present characteristics for which data were extracted (e.g., study size, PICOS, follow-up period) and provide the citations.	142-146	No. Data presented in summarized form.	0
19	Risk of bias within studies	Present data on risk of bias of each study and, if available, any outcome level assessment (see item 12).	141	No.	0
20	Results of individual studies	For all outcomes considered (benefits or harms), present, for each study: (a) simple summary data for each intervention group (b) effect estimates and confidence intervals, ideally with a forest plot.		No: study level analysis not reported.	0
21	Synthesis of results	Present results of each meta-analysis done, including confidence intervals and measures of consistency.		Results presented in concise form, preventing replication: Partial	1
22	Risk of bias across studies	Present results of any assessment of risk of bias across studies (see Item 15).		No	0
23	Additional analysis	Give results of additional analyses, if done (e.g., sensitivity or subgroup analyses, meta-regression [see Item 16]).		Results presented in concise form, preventing replication: Partial	1
<b>Discussion</b>					
24	Summary of evidence	Summarize the main findings including the strength of evidence for each main outcome; consider their relevance to key groups (e.g., healthcare providers, users, and policy makers).		Strength of evidence not presented and contextualised: Partial	1
25	Limitations	Discuss limitations at study and outcome level (e.g., risk of bias), and at review-level (e.g., incomplete retrieval of identified research, reporting bias).		No explicit discussion of study limitations	0
26	Conclusions	Provide a general interpretation of the results in the context of other evidence, and implications for future research.		Implications for future research discussed: Partial	1
<b>Funding</b>					
27	Funding	Describe sources of funding for the systematic review and other support (e.g., supply of data); role of funders for the systematic review.		No	0
<b>OVERALL SCORE</b>					<b>15</b>

# PRISMA CHECKLIST

## Scogin & McElreath, (1994)

			Page	Notes	Score
<b>Title</b>					
1	Title	Identify the report as a systematic review, meta-analysis, or both.	69	Quantitative Review/: Partial.	1
<b>Abstract</b>					
2	Structured summary	Provide a <b>structured summary</b> including, as applicable: <b>background; objectives; data sources;</b> study eligibility criteria, participants, and interventions; <b>study appraisal and synthesis methods;</b> results; <b>limitations;</b> conclusions and implications of key findings; <b>systematic review registration number.</b>		Partial	1
<b>Introduction</b>					
3	Rationale	Describe the rationale for the review in the context of what is already known.		Yes	2
4	Objectives	Provide an explicit statement of questions being addressed with reference to participants, interventions, comparisons, <b>outcomes, and study design</b> (PICOS).		Outcome and study design not explicitly included: Partial	1
<b>Methods</b>					
5	Protocol and registration	<b>Indicate if a review protocol exists, if and where it can be accessed (e.g., Web address), and, if available, provide registration information including registration number.</b>		No	0
6	Eligibility criteria	Specify study characteristics (e.g., PICOS, length of follow-up) and report characteristics (e.g., years considered, <b>language, publication status</b> ) used as criteria for eligibility, <b>giving rationale.</b>		Partial	1
7	Information sources	Describe all information sources (e.g., <b>databases with dates of coverage, contact with study authors to identify additional studies</b> ) in the search and date last searched.		computer search' not defined. Dates of coverage for selected journals noted: Partial	1
8	Search	<b>Present full electronic search strategy for at least one database, including any limits used, such that it could be repeated.</b>		No	0
9	Study selection	State the <b>process for selecting studies</b> (i.e., screening, eligibility, included in systematic review, and, if applicable, included in the meta-analysis).		Eligibility criteria described, but orices of screening not: Partial	1
10	Data collection process	Describe method of data extraction from reports (e.g., piloted forms, independently, in duplicate) <b>and any processes for obtaining and confirming data from investigators.</b>		Independent coding described: Partial	1
11	Data items	List and define all variables for which data were sought (e.g., PICOS, funding sources) and any assumptions and simplifications made.		Yes	2
12	Risk of bias in individual studies	Describe methods used for assessing risk of bias of individual studies (including specification of whether this was done at the study or outcome level), and <b>how this information is to be used in any data synthesis.</b>		Quality measure used, but not clear how this information used in data synthesis: Failsafe N used to establish impact of file-drawer bias: Partial	1
13	Summary measures	State the principal summary measures (e.g., risk ratio, difference in means).		Yes	2
14	Synthesis of results	Describe the methods of handling data and combining results of studies, <b>if done, including measures of consistency</b> (e.g., I <sup>2</sup> ) for each meta-analysis.		Yes	2
15	Risk of bias across studies	Specify any assessment of risk of bias that may affect the cumulative evidence (e.g., publication bias, <b>selective reporting within studies</b> ).		Failsafe N used to establish impact of file-drawer bias, other sources of bias not investigated: Partial	1
16	Additional analyses	<b>Describe methods of additional analyses (e.g., sensitivity or subgroup analyses, meta-regression), if done, indicating which were pre-specified</b>		Additional analyses of Quality of study length and treatment length described in results not detailed in method: No	0

PRISMA CHECKLIST		Scogin & McElreath, (1994)		
		Page	Notes	Score
<b>Results</b>				
17	Study selection		Give numbers of studies screened, assessed for eligibility, and included in the review, with reasons for exclusions at each stage, ideally with a flow diagram.	No 0
18	Study characteristics		For each study, present characteristics for which data were extracted (e.g., study size, PICOS, follow-up period) and provide the citations.	Not all data extracted (e.g. Study quality, therapist training) were given for each study. Partial 1
19	Risk of bias within studies		Present data on risk of bias of each study and, if available, any outcome level assessment (see item 12).	Risk of bias not identified for each study: Partial 1
20	Results of individual studies		For all outcomes considered (benefits or harms), present, for each study: (a) simple summary data for each intervention group (b) effect estimates and confidence intervals, ideally with a forest plot.	Partial 1
21	Synthesis of results		Present results of each meta-analysis done, including confidence intervals and measures of consistency.	Confidence Intervals not presented FS is presented: Partial 1
22	Risk of bias across studies		Present results of any assessment of risk of bias across studies (see Item 15).	No 0
23	Additional analysis		Give results of additional analyses, if done (e.g., sensitivity or subgroup analyses, meta-regression [see Item 16]).	Yes: Quality of study length and treatment length 2
<b>Discussion</b>				
24	Summary of evidence		Summarize the main findings including the strength of evidence for each main outcome; consider their relevance to key groups (e.g., healthcare providers, users, and policy makers).	strength of evidence not discussed in the context of risks of bias, except for small number of studies: Partial 1
25	Limitations		Discuss limitations at study and outcome level (e.g., risk of bias), and at review-level (e.g., incomplete retrieval of identified research, reporting bias).	Only limitation noted is small number of studies: Partial. 1
26	Conclusions		Provide a general interpretation of the results in the context of other evidence, and implications for future research.	Yes 2
<b>Funding</b>				
27	Funding		Describe sources of funding for the systematic review and other support (e.g., supply of data); role of funders for the systematic review.	No 0
<b>OVERALL SCORE</b>				<b>27</b>

# PRISMA CHECKLIST

## Engels & Vermey (1997)

			Page	Notes	Score
<b>Title</b>					
1	Title	Identify the report as a systematic review, meta-analysis, or both.	17	Quantitative Analysis': Partial.	1
<b>Abstract</b>					
2	Structured summary	Provide a <b>structured summary</b> including, as applicable: <b>background</b> ; <b>objectives</b> ; <b>data sources</b> ; <b>study eligibility criteria, participants, and interventions</b> ; <b>study appraisal and synthesis methods</b> ; <b>results</b> ; <b>limitations</b> ; <b>conclusions and implications of key findings</b> ; <b>systematic review registration number</b> .	17	Partial	1
<b>Introduction</b>					
3	Rationale	Describe the rationale for the review in the context of what is already known.	18	yes	2
4	Objectives	Provide an explicit statement of questions being addressed with reference to participants, interventions, comparisons, outcomes, and <b>study design (PICOS)</b> .	18	Study design not explicitly included in objectives: partial	1
<b>Methods</b>					
5	Protocol and registration	<b>Indicate if a review protocol exists, if and where it can be accessed (e.g., Web address), and, if available, provide registration information including registration number.</b>	19	no	0
6	Eligibility criteria	Specify study characteristics (e.g., PICOS, length of follow-up) and report characteristics (e.g., years considered, <b>language, publication status</b> ) used as criteria for eligibility, <b>giving rationale</b> .	19	Partial	1
7	Information sources	Describe all information sources (e.g., databases with dates of coverage, <b>contact with study authors to identify additional studies</b> ) in the search and <b>date last searched</b> .	19	Partial	1
8	Search	<b>Present full electronic search strategy for at least one database, including any limits used, such that it could be repeated.</b>	19	No	0
9	Study selection	<b>State the process for selecting studies (i.e., screening, eligibility, included in systematic review, and, if applicable, included in the meta-analysis).</b>	19	Screening process not outlined systematically: Partial	1
10	Data collection process	<b>Describe method of data extraction from reports (e.g., piloted forms, independently, in duplicate) and any processes for obtaining and confirming data from investigators.</b>	19	Mean inter-rater agreement reported. No communication with authors reported: Partial.	1
11	Data items	List and define all variables for which data were sought (e.g., PICOS, funding sources) and any assumptions and simplifications made.	21-22	Yes	2
12	Risk of bias in individual studies	<b>Describe methods used for assessing risk of bias of individual studies (including specification of whether this was done at the study or outcome level), and how this information is to be used in any data synthesis.</b>	22	No	0
13	Summary measures	State the principal summary measures (e.g., risk ratio, difference in means).	23	Yes	2
14	Synthesis of results	Describe the methods of handling data and combining results of studies, <b>if done, including measures of consistency (e.g., I<sup>2</sup>) for each meta-analysis.</b>	22-23	Methods outlined, but not detailed specifically for ind studies: Partial	1
15	Risk of bias across studies	<b>Specify any assessment of risk of bias that may affect the cumulative evidence (e.g., publication bias, selective reporting within studies).</b>	23	no	0
16	Additional analyses	<b>Describe methods of additional analyses (e.g., sensitivity or subgroup analyses, meta-regression), if done, indicating which were pre-specified</b>	23	Sub-group analysis not described explicitly	0

PRISMA CHECKLIST		Engels & Vermeij (1997)		Score	
		Page	Notes		
<b>Results</b>					
17	Study selection	Give numbers of studies screened, assessed for eligibility, and included in the review, with reasons for exclusions at each stage, ideally with a flow diagram.	23	Pre-screening numbers and reasons for exclusion not given: Partial	1
18	Study characteristics	For each study, present characteristics for which data were extracted (e.g., study size, PICOS, follow-up period) and provide the citations.	25	No	0
19	Risk of bias within studies	Present data on risk of bias of each study and, if available, any outcome level assessment (see item 12).	29	Internal validity of studies explored: assignment, drop-out, reporting consistency, Not systematic for each study: Partial	1
20	Results of individual studies	For all outcomes considered (benefits or harms), present, for each study: (a) simple summary data for each intervention group (b) effect estimates and confidence intervals, ideally with a forest plot.	29	No	0
21	Synthesis of results	Present results of each meta-analysis done, including confidence intervals and measures of consistency.	24-28	CI and consistency not reported: Partial	1
22	Risk of bias across studies	Present results of any assessment of risk of bias across studies (see Item 15).	30	Publication bias funnel plot included, other factors not detailed: Partial.	1
23	Additional analysis	Give results of additional analyses, if done (e.g., sensitivity or subgroup analyses, meta-regression [see Item 16]).	24-28	Yes: Sub-group analysis part of main findings: Mean effect sizes reported.	2
<b>Discussion</b>					
24	Summary of evidence	Summarize the main findings including the strength of evidence for each main outcome; consider their relevance to key groups (e.g., healthcare providers, users, and policy makers).	30-33	Relevance to key groups not explored: Partial.	1
25	Limitations	Discuss limitations at study and outcome level (e.g., risk of bias), and at review-level (e.g., incomplete retrieval of identified research, reporting bias).	33	Yes	2
26	Conclusions	Provide a general interpretation of the results in the context of other evidence, and implications for future research.	33	Yes	2
<b>Funding</b>					
27	Funding	Describe sources of funding for the systematic review and other support (e.g., supply of data); role of funders for the systematic review.	33	Acknowledgements given: part-fulfillment of masters degree, all sources of funding not detailed: Partial.	1
<b>OVERALL SCORE</b>				<b>26</b>	

# PRISMA CHECKLIST

## Cuijpers (1998)

			Page	Notes	Score
<b>Title</b>					
1	Title	Identify the report as a systematic review, meta-analysis, or both.	41	Yes	2
<b>Abstract</b>					
2	Structured summary	Provide a <b>structured summary</b> including, as applicable: background; objectives; <b>data sources</b> ; study eligibility criteria, participants, and interventions; study appraisal and synthesis methods; results; <b>limitations</b> ; conclusions and implications of key findings; systematic review registration number.	41	Data source not noted: MA and meta-regression analysis methods mentioned, not detailed. Limitations not described. Partial	1
<b>Introduction</b>					
3	Rationale	Describe the rationale for the review in the context of what is already known.	41	Yes	2
4	Objectives	Provide an explicit statement of questions being addressed with reference to participants, interventions, comparisons, <b>outcomes</b> , and <b>study design</b> (PICOS).	41	Yes: depressed OA, psychotherapy, active recruitment, outreach, outcomes measured and study design not indicated: Partial	1
<b>Methods</b>					
5	Protocol and registration	<b>Indicate if a review protocol exists, if and where it can be accessed (e.g., Web address), and, if available, provide registration information including registration number.</b>	42	No	0
6	Eligibility criteria	Specify study characteristics (e.g., PICOS, length of follow-up) and report characteristics (e.g., <b>years considered, language, publication status</b> ) used as criteria for eligibility, <b>giving rationale.</b>	42	Partial	1
7	Information sources	Describe all information sources (e.g., databases with <b>dates of coverage, contact with study authors to identify additional studies in the search and date last searched.</b>	42	Partial	1
8	Search	<b>Present full electronic search strategy for at least one database, including any limits used, such that it could be repeated.</b>	42	No	0
9	Study selection	State the <b>process</b> for selecting studies (i.e., screening, eligibility, included in systematic review, and, if applicable, included in the meta-analysis).	42	Discussion of eligibility of studies with 'psychotherapeutic arms' outlined. Process not described: Partial	1
10	Data collection process	<b>Describe method of data extraction from reports (e.g., piloted forms, independently, in duplicate) and any processes for obtaining and confirming data from investigators.</b>	42	Data collection process not outlined (independent or not, contact with authors etc.): No	0
11	Data items	List and define all variables for which data were sought (e.g., PICOS, funding sources) and any assumptions and simplifications made.	42	Yes	2
12	Risk of bias in individual studies	<b>Describe methods used for assessing risk of bias of individual studies (including specification of whether this was done at the study or outcome level), and how this information is to be used in any data synthesis.</b>	42	No	0
13	Summary measures	State the principal summary measures (e.g., risk ratio, difference in means).	43	yes: dropout, effect sizes	2
14	Synthesis of results	Describe the methods of handling data and combining results of studies, if done, including <b>measures of consistency</b> (e.g., I <sup>2</sup> ) for each meta-analysis.	44	methods used are well-described.	2
15	Risk of bias across studies	<b>Specify any assessment of risk of bias that may affect the cumulative evidence (e.g., publication bias, selective reporting within studies).</b>	44	No explicit discussion of risk of bias: but WL control and small sample were noted as limiting factors: partial.	1
16	Additional analyses	Describe methods of additional analyses (e.g., sensitivity or subgroup analyses, meta-regression), if done, indicating which were pre-specified	44	Yes: Analysis of drop-out was detailed and identified in original objectives.	2

PRISMA CHECKLIST		Cuijpers, (1998)		Score		
		Page	Notes			
<b>Results</b>						
17	Study selection		Give numbers of studies screened, assessed for eligibility, and included in the review, with reasons for exclusions at each stage, ideally with a flow diagram.	No	0	
18	Study characteristics		For each study, present characteristics for which data were extracted (e.g., study size, PICOS, follow-up period) and provide the citations.	43	yes	2
19	Risk of bias within studies		Present data on risk of bias of each study and, if available, any outcome level assessment (see item 12).	44	no	0
20	Results of individual studies		For all outcomes considered (benefits or harms), present, for each study: (a) simple summary data for each intervention group (b) effect estimates and confidence intervals, ideally with a forest plot.		Summary table identified summary data and effect sizes, but CI only reported for mean: Partial	1
21	Synthesis of results		Present results of each meta-analysis done, including confidence intervals and measures of consistency.		yes	2
22	Risk of bias across studies		Present results of any assessment of risk of bias across studies (see Item 15).		No	0
23	Additional analysis		Give results of additional analyses, if done (e.g., sensitivity or subgroup analyses, meta-regression [see Item 16]).		Analysis of drop-out and multiple regression analysis reported	2
<b>Discussion</b>						
24	Summary of evidence		Summarize the main findings including the strength of evidence for each main outcome; consider their relevance to key groups (e.g., healthcare providers, users, and policy makers).		The reported effect size is not contextualised within the potential impact of limiting factors, despite some being acknowledged: Partial	1
25	Limitations		Discuss limitations at study and outcome level (e.g., risk of bias), and at review-level (e.g., incomplete retrieval of identified research, reporting bias).		review level risk of bias not discussed: Partial	1
26	Conclusions		Provide a general interpretation of the results in the context of other evidence, and implications for future research.		Yes	2
<b>Funding</b>						
27	Funding		Describe sources of funding for the systematic review and other support (e.g., supply of data); role of funders for the systematic review.		No	0
<b>OVERALL SCORE</b>					<b>29</b>	



# PRISMA CHECKLIST

# Pinquart & Sorensen (2001)

			Page	Notes	Score
<b>Title</b>					
1	Title	Identify the report as a systematic review, meta-analysis, or both.	207	Yes	2
<b>Abstract</b>					
2	Structured summary	Provide a structured summary including, as applicable: background; objectives; data sources; study eligibility criteria, participants, and interventions; study appraisal and synthesis methods; results; limitations; conclusions and implications of key findings; systematic review registration number.	207	Summary of results: No details of methodology, implications or limitations: Partial.	1
<b>Introduction</b>					
3	Rationale	Describe the rationale for the review in the context of what is already known.	208	Yes	2
4	Objectives	Provide an explicit statement of questions being addressed with reference to participants, interventions, comparisons, outcomes, and study design (PICOS).	213-214	Yes	2
<b>Methods</b>					
5	Protocol and registration	Indicate if a review protocol exists, if and where it can be accessed (e.g., Web address), and, if available, provide registration information including registration number.	214	No	0
6	Eligibility criteria	Specify study characteristics (e.g., PICOS, length of follow-up) and report characteristics (e.g., years considered, language, publication status) used as criteria for eligibility, giving rationale.	214-215	No information on years considered, publication status: Partial	1
7	Information sources	Describe all information sources (e.g., databases with dates of coverage, contact with study authors to identify additional studies) in the search and date last searched.	214	Not described systematically. Partial.	1
8	Search	Present full electronic search strategy for at least one database, including any limits used, such that it could be repeated.	214	No	0
9	Study selection	State the process for selecting studies (i.e., screening, eligibility, included in systematic review, and, if applicable, included in the meta-analysis).	214	Process not described. No	0
10	Data collection process	Describe method of data extraction from reports (e.g., piloted forms, independently, in duplicate) and any processes for obtaining and confirming data from investigators.	215	No	0
11	Data items	List and define all variables for which data were sought (e.g., PICOS, funding sources) and any assumptions and simplifications made.	215	Yes	2
12	Risk of bias in individual studies	Describe methods used for assessing risk of bias of individual studies (including specification of whether this was done at the study or outcome level), and how this information is to be used in any data synthesis.	215	Quality coded. Effect sizes adjusted for biases due to differences in pre-test scores and over-estimation of popln ES: Partial	1
13	Summary measures	State the principal summary measures (e.g., risk ratio, difference in means).	215-216	Yes	2
14	Synthesis of results	Describe the methods of handling data and combining results of studies, if done, including measures of consistency (e.g., I <sup>2</sup> ) for each meta-analysis.	215-216	Yes, homogeneity of ES calculated.	2
15	Risk of bias across studies	Specify any assessment of risk of bias that may affect the cumulative evidence (e.g., publication bias, selective reporting within studies).	215	No	0
16	Additional analyses	Describe methods of additional analyses (e.g., sensitivity or subgroup analyses, meta-regression), if done, indicating which were pre-specified	216	methods of multiple linear regressions to analyse moderator effects noted but not described: Partial	1



PRISMA CHECKLIST		Pinquart & Sorensen (2001)			
		Page	Notes	Score	
<b>Results</b>					
17	Study selection	Give numbers of studies screened, assessed for eligibility, and included in the review, with reasons for exclusions at each stage, ideally with a flow diagram.	216	No	0
18	Study characteristics	For each study, present characteristics for which data were extracted (e.g., study size, PICOS, follow-up period) and provide the citations.	216	No	0
19	Risk of bias within studies	Present data on risk of bias of each study and, if available, any outcome level assessment (see item 12).	216	No	0
20	Results of individual studies	For all outcomes considered (benefits or harms), present, for each study: (a) simple summary data for each intervention group (b) effect estimates and confidence intervals, ideally with a forest plot.		No. Large number of studies: reported only synthesis.	0
21	Synthesis of results	Present results of each meta-analysis done, including confidence intervals and measures of consistency.	218-219	Yes	2
22	Risk of bias across studies	Present results of any assessment of risk of bias across studies (see Item 15).	226	Influence of Quality rating of studies on ES calculated: Partial	1
23	Additional analysis	Give results of additional analyses, if done (e.g., sensitivity or subgroup analyses, meta-regression [see Item 16]).	222-225	Yes	2
<b>Discussion</b>					
24	Summary of evidence	Summarize the main findings including the strength of evidence for each main outcome; consider their relevance to key groups (e.g., healthcare providers, users, and policymakers).	225-231	Yes	2
25	Limitations	Discuss limitations at study and outcome level (e.g., risk of bias), and at review-level (e.g., incomplete retrieval of identified research, reporting bias).	230	Yes	2
26	Conclusions	Provide a general interpretation of the results in the context of other evidence, and implications for future research.	225-231	Yes	2
<b>Funding</b>					
27	Funding	Describe sources of funding for the systematic review and other support (e.g., supply of data); role of funders for the systematic review.	231	No	0
<b>OVERALL SCORE</b>					<b>28</b>

## PRISMA CHECKLIST

Bohlmeijer *et al.*, (2003)

			Page	Notes	Score
<b>Title</b>					
1	Title	Identify the report as a systematic review, meta-analysis, or both.	1088	yes	2
<b>Abstract</b>					
2	Structured summary	Provide a structured summary including, as applicable: <b>background</b> ; objectives; data sources; <b>study eligibility criteria</b> , participants, and interventions; <b>study appraisal</b> and synthesis methods; results; <b>limitations</b> ; conclusions and implications of key findings; <b>systematic review registration number</b> .	1088	Partial	1
<b>Introduction</b>					
3	Rationale	Describe the rationale for the review in the context of what is already known.	1089	Yes	2
4	Objectives	Provide an <b>explicit statement of questions being addressed</b> with reference to participants, interventions, comparisons, outcomes, and study design (PICOS).	1089	Explicit questions not described: PICOS factors are all described, except study design: Partial.	1
<b>Methods</b>					
5	Protocol and registration	<b>Indicate if a review protocol exists, if and where it can be accessed (e.g., Web address), and, if available, provide registration information including registration number.</b>	1089	No	0
6	Eligibility criteria	Specify study characteristics (e.g., PICOS, length of follow-up) and report characteristics (e.g., years considered, language, publication status) used as criteria for eligibility, giving rationale.	1089	Yes	2
7	Information sources	Describe all information sources (e.g., databases with dates of coverage, <b>contact with study authors to identify additional studies</b> ) in the search and <b>date last searched</b> .	1089	Partial	1
8	Search	<b>Present full electronic search strategy for at least one database, including any limits used, such that it could be repeated.</b>	1089	No	0
9	Study selection	State the <b>process for selecting studies</b> (i.e., screening, eligibility, included in systematic review, and, if applicable, included in the meta-analysis).	1089	process not described, but eligibility outlined: Partial	1
10	Data collection process	Describe method of data extraction from reports (e.g., piloted forms, independently, in duplicate) and any processes for obtaining and confirming data from investigators.	1089	No. Method not outlined	0
11	Data items	List and <b>define all variables for which data were sought</b> (e.g., PICOS, funding sources) and any assumptions and simplifications made.	1089	Variables listed, but not defined: (e.g. Study quality). Partial	1
12	Risk of bias in individual studies	<b>Describe methods used for assessing risk of bias of individual studies (including specification of whether this was done at the study or outcome level), and how this information is to be used in any data synthesis.</b>	1090	Study quality was not reported for each included study, and risk of bias not explicitly discussed or its impact of synthesis.	0
13	Summary measures	State the principal summary measures (e.g., risk ratio, difference in means).	1091	Yes: ES	2
14	Synthesis of results	Describe the methods of handling data and combining results of studies, if done, <b>including measures of consistency</b> (e.g., I <sup>2</sup> ) for each meta-analysis.	1091	MA method described. No measures of consistency	2
15	Risk of bias across studies	Specify any assessment of risk of bias that may affect the cumulative evidence (e.g., publication bias, <b>selective reporting within studies</b> ).	1091	Publication bias considered. Correction for the reliability (Cronbachs $\alpha$ or test-retest reliability $r$ ): Partial.	1
16	Additional analyses	<b>Describe methods of additional analyses (e.g., sensitivity or subgroup analyses, meta-regression), if done, indicating which were pre-specified</b>	1091	No. Although sub-group analyses undertaken, not outlined in methods.	0

PRISMA CHECKLIST		Bohlmeijer et al., (2003)			
		Page	Notes	Score	
<b>Results</b>					
17	Study selection		Give numbers of studies screened, assessed for eligibility, and included in the review, with reasons for exclusions at each stage, ideally with a flow diagram.	No	0
18	Study characteristics	1090	For each study, present characteristics for which data were extracted (e.g., study size, PICOS, follow-up period) and provide the citations.	Yes	2
19	Risk of bias within studies	1090-1091	Present data on risk of bias of each study and, if available, any outcome level assessment (see item 12).	No, although risk of therapeutic alliance identified for one trial.	0
20	Results of individual studies	1090-1091	For all outcomes considered (benefits or harms), present, for each study: (a) simple summary data for each intervention group (b) effect estimates and confidence intervals, ideally with a forest plot.	No	0
21	Synthesis of results	1092	Present results of each meta-analysis done, including confidence intervals and measures of consistency.	Yes	2
22	Risk of bias across studies	1092	Present results of any assessment of risk of bias across studies (see Item 15).	No	0
23	Additional analysis	1092	Give results of additional analyses, if done (e.g., sensitivity or subgroup analyses, meta-regression [see Item 16]).	Yes	2
<b>Discussion</b>					
24	Summary of evidence	1093	Summarize the main findings including the strength of evidence for each main outcome; consider their relevance to key groups (e.g., healthcare providers, users, and policymakers).	Yes	2
25	Limitations	1092	Discuss limitations at study and outcome level (e.g., risk of bias), and at review-level (e.g., incomplete retrieval of identified research, reporting bias).	Partial	1
26	Conclusions	1093	Provide a general interpretation of the results in the context of other evidence, and implications for future research.	Yes	2
<b>Funding</b>					
27	Funding	109	Describe sources of funding for the systematic review and other support (e.g., supply of data); role of funders for the systematic review.	No	0
<b>OVERALL SCORE</b>					<b>27</b>

# PRISMA CHECKLIST

Pinquart *et al.*, (2006)

			Page	Notes	Score
<b>Title</b>					
1	Title	Identify the report as a systematic review, meta-analysis, or both.	1493	Yes	2
<b>Abstract</b>					
2	Structured summary	Provide a structured summary including, as applicable: background; objectives; data sources; study eligibility criteria, participants, and interventions; study appraisal and synthesis methods; results; limitations; conclusions and implications of key findings; <b>systematic review registration number</b> .	1493	detailed structured summary, only missing 2 review registration.	2
<b>Introduction</b>					
3	Rationale	Describe the rationale for the review in the context of what is already known.	1493	Yes	2
4	Objectives	Provide an explicit statement of questions being addressed with reference to participants, interventions, comparisons, outcomes, and study design (PICOS).	1494	Yes	2
<b>Methods</b>					
5	Protocol and registration	<b>Indicate if a review protocol exists, if and where it can be accessed (e.g., Web address), and, if available, provide registration information including registration number.</b>	1494	No	0
6	Eligibility criteria	Specify study characteristics (e.g., PICOS, length of follow-up) and report characteristics (e.g., <b>years considered</b> , language, <b>publication status</b> ) used as criteria for eligibility, giving rationale.	1494-1495	Date of studies, and published/non-published not defined: Partial	1
7	Information sources	Describe all information sources (e.g., databases with <b>dates of coverage, contact with study authors to identify additional studies</b> ) in the search and date last searched.	1494-1495	Partial	1
8	Search	<b>Present full electronic search strategy for at least one database, including any limits used, such that it could be repeated.</b>	1494-1495	No	0
9	Study selection	State the process for selecting studies (i.e., screening, eligibility, included in systematic review, and, if applicable, included in the meta-analysis).	1495	Details of reasons for exclusion given, but details screening at title/abstract/full-text not given: Partial.	1
10	Data collection process	Describe method of data extraction from reports (e.g., piloted forms, independently, in duplicate) and <b>any processes for obtaining and confirming data from investigators.</b>	1495	Yes: two coders (Cohen's kappa) of 0.87 for 20% sample. Investigators not contacted; Partial.	1
11	Data items	List and define all variables for which data were sought (e.g., PICOS, funding sources) and any assumptions and simplifications made.	1495	Yes	2
12	Risk of bias in individual studies	Describe methods used for assessing risk of bias of individual studies (including specification of whether this was done at the study or outcome level), and <b>how this information is to be used in any data synthesis.</b>	1495	Quality assessment used, but not clear how this information informed data synthesis: Effect size estimates adjusted for bias due to baseline differences in depression scores. Partial	1
13	Summary measures	State the principal summary measures (e.g., risk ratio, difference in means).	1497	Yes	2
14	Synthesis of results	Describe the methods of handling data and combining results of studies, if done, including measures of consistency (e.g., I <sup>2</sup> ) for each meta-analysis.	1498	Yes: ES with 95% CI given with Homogeneity of ES (Q), (but not I <sup>2</sup> )	2
15	Risk of bias across studies	Specify any assessment of risk of bias that may affect the cumulative evidence (e.g., publication bias, selective reporting within studies).	1495	Effect size estimates adjusted for bias due to baseline differences in depression scores: Partial	1
16	Additional analyses	Describe methods of additional analyses (e.g., sensitivity or subgroup analyses, meta-regression), <b>if done, indicating which were pre-specified</b>	1498	Reported in Results: not identified as pre-specified in Methods: No	0

PRISMA CHECKLIST		Pinquart et al., (2006)		Score	
		Page	Notes		
<b>Results</b>					
17	Study selection	Give numbers of studies screened, assessed for eligibility, and included in the review, with reasons for exclusions at each stage, ideally with a flow diagram.	1498	No.	0
18	Study characteristics	For each study, present characteristics for which data were extracted (e.g., study size, PICOS, follow-up period) and provide the citations.	1495	Link to web-based list of eligible studies no longer worked;No	0
19	Risk of bias within studies	Present data on risk of bias of each study and, if available, any outcome level assessment (see item 12).	1498	No	0
20	Results of individual studies	For all outcomes considered (benefits or harms), present, for each study: (a) simple summary data for each intervention group (b) effect estimates and confidence intervals, ideally with a forest plot.	1498	No	0
21	Synthesis of results	Present results of each meta-analysis done, including confidence intervals and measures of consistency.	1497-1498	Yes	2
22	Risk of bias across studies	Present results of any assessment of risk of bias across studies (see Item 15).	1498	No	0
23	Additional analysis	Give results of additional analyses, if done (e.g., sensitivity or subgroup analyses, meta-regression [see Item 16]).	1498	yes	2
<b>Discussion</b>					
24	Summary of evidence	Summarize the main findings including the strength of evidence for each main outcome; consider their relevance to key groups (e.g., healthcare providers, users, and policymakers).	1499	Yes	2
25	Limitations	Discuss limitations at study and outcome level (e.g., risk of bias), and at review-level (e.g., incomplete retrieval of identified research, reporting bias).	1499-1500	Yes	2
26	Conclusions	Provide a general interpretation of the results in the context of other evidence, and implications for future research.	1500	Yes	2
<b>Funding</b>					
27	Funding	Describe sources of funding for the systematic review and other support (e.g., supply of data); role of funders for the systematic review.	1500	Yes	2

OVERALL SCORE

32

## PRISMA CHECKLIST

Cuijpers *et al.*, (2006)

			Page	Notes	Score
<b>Title</b>					
1	Title	Identify the report as a systematic review, meta-analysis, or both.	1139	Yes	2
<b>Abstract</b>					
2	Structured summary	Provide a structured summary including, as applicable: background; objectives; <b>data sources</b> ; study eligibility criteria, participants, and interventions; <b>study appraisal and synthesis methods</b> ; results; limitations; conclusions and implications of key findings; <b>systematic review registration number</b> .	1139	Partial	1
<b>Introduction</b>					
3	Rationale	Describe the rationale for the review in the context of what is already known.	1139-1140	Yes	2
4	Objectives	Provide an explicit statement of questions being addressed with reference to participants, interventions, comparisons, outcomes, and study design (PICOS).	1140	Yes	2
<b>Methods</b>					
5	Protocol and registration	<b>Indicate if a review protocol exists, if and where it can be accessed (e.g., Web address), and, if available, provide registration information including registration number.</b>	1140	No	0
6	Eligibility criteria	Specify study characteristics (e.g., PICOS, length of follow-up) and report characteristics (e.g., years considered, language, publication status) used as criteria for eligibility, giving rationale.	1140	Yes	2
7	Information sources	Describe all information sources (e.g., databases with dates of coverage, <b>contact with study authors to identify additional studies</b> ) in the search and <b>date last searched</b> .	1140	Partial	1
8	Search	<b>Present full electronic search strategy for at least one database, including any limits used, such that it could be repeated.</b>	1140	No	0
9	Study selection	State the <b>process</b> for selecting studies (i.e., screening, eligibility, included in systematic review, and, if applicable, included in the meta-analysis).	1140	process of screening not fully reported: e.g. abstracts/full paper: Partial	1
10	Data collection process	<b>Describe method of data extraction from reports (e.g., piloted forms, independently, in duplicate) and any processes for obtaining and confirming data from investigators.</b>	1140	No	0
11	Data items	List and define all variables for which data were sought (e.g., PICOS, funding sources) and any assumptions and simplifications made.	1140	Yes	2
12	Risk of bias in individual studies	<b>Describe methods used for assessing risk of bias of individual studies (including specification of whether this was done at the study or outcome level), and how this information is to be used in any data synthesis.</b>	1143	No	0
13	Summary measures	State the principal summary measures (e.g., risk ratio, difference in means).	1143	Yes	2
14	Synthesis of results	Describe the methods of handling data and combining results of studies, if done, including measures of consistency (e.g., I <sup>2</sup> ) for each meta-analysis.	1143	Yes	2
15	Risk of bias across studies	<b>Specify any assessment of risk of bias that may affect the cumulative evidence (e.g., publication bias, selective reporting within studies).</b>	1140-1143	No	0
16	Additional analyses	Describe methods of additional analyses (e.g., sensitivity or subgroup analyses, meta-regression), if done, <b>indicating which were pre-specified</b>	1143	Pre-specification of regression analysis not noted: Partial	1

PRISMA CHECKLIST			Cuijpers et al., (2006)		
			Page	Notes	Score
<b>Results</b>					
17	Study selection	Give numbers of studies screened, assessed for eligibility, and included in the review, with reasons for exclusions at each stage, ideally with a flow diagram.	1143	Although no flow diagram, other criteria met.	2
18	Study characteristics	For each study, present characteristics for which data were extracted (e.g., study size, PICOS, follow-up period) and provide the citations.	1141-1143	Yes	2
19	Risk of bias within studies	Present data on risk of bias of each study and, if available, any outcome level assessment (see item 12).	1144	Although risk of bias not explicitly discussed, randomisation and blinding detailed: Partial	1
20	Results of individual studies	For all outcomes considered (benefits or harms), present, for each study: (a) simple summary data for each intervention group (b) effect estimates and confidence intervals, ideally with a forest plot.	1145	yes	2
21	Synthesis of results	Present results of each meta-analysis done, including confidence intervals and measures of consistency.	1144	Yes	2
22	Risk of bias across studies	Present results of any assessment of risk of bias across studies (see Item 15).	1145-46	No	0
23	Additional analysis	Give results of additional analyses, if done (e.g., sensitivity or subgroup analyses, meta-regression [see Item 16]).	1146	Yes	2
<b>Discussion</b>					
24	Summary of evidence	Summarize the main findings including the strength of evidence for each main outcome; consider their relevance to key groups (e.g., healthcare providers, users, and policy makers).	1146-7	Yes	2
25	Limitations	Discuss limitations at study and outcome level (e.g., risk of bias), and at review-level (e.g., incomplete retrieval of identified research, reporting bias).	1147	Limitations of both study quality and review-methodology are discussed e.g. Exclusion of studies to get heterogeneous sample. Yes	2
26	Conclusions	Provide a general interpretation of the results in the context of other evidence, and implications for future research.	1147	Yes	2
<b>Funding</b>					
27	Funding	Describe sources of funding for the systematic review and other support (e.g., supply of data); role of funders for the systematic review.		No	0
<b>OVERALL SCORE</b>					<b>35</b>



# PRISMA CHECKLIST

## Chin (2007)

			Page	Notes	Score
<b>Title</b>					
1	Title	Identify the report as a systematic review, meta-analysis, or both.	10	Yes	2
<b>Abstract</b>					
2	Structured summary	Provide a structured summary including, as applicable: <b>background</b> ; objectives; data sources; study eligibility criteria, participants, and interventions; study appraisal and synthesis methods; results; limitations; conclusions and implications of key findings; <b>systematic review registration number</b> .	10	Structured abstract detailing all but background.	2
<b>Introduction</b>					
3	Rationale	Describe the rationale for the review in the context of what is already known.	11	Yes	2
4	Objectives	Provide an explicit statement of questions being addressed with reference to participants, interventions, <b>comparisons</b> , outcomes, and study design (PICOS).		Comparison groups to be examined not outlined in objectives: Partial	1
<b>Methods</b>					
5	Protocol and registration	<b>Indicate if a review protocol exists, if and where it can be accessed (e.g., Web address), and, if available, provide registration information including registration number.</b>	11	No	0
6	Eligibility criteria	Specify study characteristics (e.g., PICOS, length of follow-up) and report characteristics (e.g., years considered, <b>language, publication status</b> ) used as criteria for eligibility, giving rationale.	11	Publication status, language not identified. Rationale for not requiring blinding given: Partial	1
7	Information sources	Describe all information sources (e.g., databases with dates of coverage, contact with study authors to identify additional studies) in the search and <b>date last searched</b> .	12	Date of last search not given: Partial	1
8	Search	<b>Present full electronic search strategy for at least one database, including any limits used, such that it could be repeated.</b>	12	No	0
9	Study selection	State the process for selecting studies (i.e., screening, eligibility, included in systematic review, and, if applicable, included in the meta-analysis).	12	Yes	2
10	Data collection process	Describe method of data extraction from reports (e.g., piloted forms, independently, in duplicate) and any processes for obtaining and confirming data from investigators.	12	Yes	2
11	Data items	List and define all variables for which data were sought (e.g., PICOS, funding sources) and any assumptions and simplifications made.	12	Yes	2
12	Risk of bias in individual studies	Describe methods used for assessing risk of bias of individual studies ( <b>including specification of whether this was done at the study or outcome level</b> ), and <b>how this information is to be used in any data synthesis</b> .	12	quality assessment using generic scale: Yes	1
13	Summary measures	State the principal summary measures (e.g., risk ratio, difference in means).	12	Not explicitly, but details included in 'Data analysis': Partial	1
14	Synthesis of results	Describe the methods of handling data and combining results of studies, if done, including measures of consistency (e.g., I <sup>2</sup> ) for each meta-analysis.	12-13	Yes	2
15	Risk of bias across studies	Specify any assessment of risk of bias that may affect the cumulative evidence (e.g., publication bias, selective reporting within studies).	13	Failsafe-N method outlined. Sensitivity Analysis undertaken. Rationale for not undertaking sub-group analysis given: Yes	2
16	Additional analyses	<b>Describe methods of additional analyses (e.g., sensitivity or subgroup analyses, meta-regression), if done, indicating which were pre-specified</b>	13	Rationale for not undertaking sub-group analysis given: but not undertaken.	0



PRISMA CHECKLIST		Chin (2007)		Score	
		Page	Notes		
<b>Results</b>					
17	Study selection	Give numbers of studies screened, assessed for eligibility, and included in the review, with reasons for exclusions at each stage, ideally with a flow diagram.	13	Total studies screened not listed, no flow diagram, but reasons for exclusion of those after screening title/abstract given: Partial	1
18	Study characteristics	For each study, present characteristics for which data were extracted (e.g., study size, PICOS, follow-up period) and provide the citations.	14-17	Yes: Across categories of analysis.	2
19	Risk of bias within studies	Present data on risk of bias of each study and, if available, any outcome level assessment (see item 12).	14-17	Partial: assignment, blinding of outcome, attrition rates extracted in addition to quality assessment using generic scale	1
20	Results of individual studies	For all outcomes considered (benefits or harms), present, for each study: (a) simple summary data for each intervention group (b) effect estimates and confidence intervals, ideally with a forest plot.	18-20	Organised according to outcome measures used: summary data for each study therefore split across a number of tables/forest plots, but information extractable. Yes	2
21	Synthesis of results	Present results of each meta-analysis done, including confidence intervals and measures of consistency.	18-20	Yes, although Q or I2 statistic of consistency not done: Partial	1
22	Risk of bias across studies	Present results of any assessment of risk of bias across studies (see Item 15).	15-16	File drawer analysis reported as was analysis of skew with correction: Yes	2
23	Additional analysis	Give results of additional analyses, if done (e.g., sensitivity or subgroup analyses, meta-regression [see Item 16]).	15-18	No meta-regression/ sub-group undertaken.	0
<b>Discussion</b>					
24	Summary of evidence	Summarize the main findings including the strength of evidence for each main outcome; consider their relevance to key groups (e.g., healthcare providers, users, and policy makers).	19-20	Summary, but partial.	1
25	Limitations	Discuss limitations at study and outcome level (e.g., risk of bias), and at review-level (e.g., incomplete retrieval of identified research, reporting bias).	21	Yes	2
26	Conclusions	Provide a general interpretation of the results in the context of other evidence, and implications for future research.	21	Yes, but not in context of other research: Partial	1
<b>Funding</b>					
27	Funding	Describe sources of funding for the systematic review and other support (e.g., supply of data); role of funders for the systematic review.	21	No	0
<b>OVERALL SCORE</b>					<b>34</b>

## PRISMA CHECKLIST

Pinquart *et al.*, (2007)

			Page	Notes	Score
<b>Title</b>					
1	Title	Identify the report as a systematic review, meta-analysis, or both.	645	Yes	2
<b>Abstract</b>					
2	Structured summary	Provide a structured summary including, as applicable: <b>background</b> ; objectives; <b>data sources</b> ; <b>study eligibility criteria, participants</b> , and interventions; <b>study appraisal and synthesis methods</b> ; results; limitations; conclusions and implications of key findings; systematic review registration number.	645	Partial	1
<b>Introduction</b>					
3	Rationale	Describe the rationale for the review in the context of what is already known.	645-647	Yes	2
4	Objectives	Provide an <b>explicit statement of questions</b> being addressed with reference to participants, interventions, comparisons, outcomes, and study design (PICOS).	647-648	Yes. Explicit expectations across PICOS outlined, although not in question form, specificity of research questions are clear.	2
<b>Methods</b>					
5	Protocol and registration	<b>Indicate if a review protocol exists, if and where it can be accessed (e.g., Web address), and, if available, provide registration information including registration number.</b>	648	No	0
6	Eligibility criteria	Specify study characteristics (e.g., PICOS, length of follow-up) and report characteristics (e.g., years considered, language, <b>publication status</b> ) used as criteria for eligibility, <b>giving rationale.</b>	648	Partial	1
7	Information sources	Describe all information sources (e.g., databases with dates of coverage, <b>contact with study authors to identify additional studies</b> ) in the search and <b>date last searched.</b>	648	Partial	1
8	Search	<b>Present full electronic search strategy for at least one database, including any limits used, such that it could be repeated.</b>	648	No	0
9	Study selection	State the <b>process for selecting studies</b> (i.e., screening, eligibility, included in systematic review, and, if applicable, included in the meta-analysis).	648	No: description of process of selection not given	0
10	Data collection process	<b>Describe method of data extraction from reports</b> (e.g., piloted forms, independently, in duplicate) and <b>any processes for obtaining and confirming data from investigators.</b>	648	Inter-rater agreement $k = .09$ for coding reported: Partial	1
11	Data items	List and define all variables for which data were sought (e.g., PICOS, funding sources) and any assumptions and simplifications made.	648	Yes	2
12	Risk of bias in individual studies	Describe methods used for assessing risk of bias of individual studies (including specification of whether this was done at the study or outcome level), <b>and how this information is to be used in any data synthesis.</b>	648	Quality criteria described, without explicit discussion of study level bias. Effect size estimates adjusted for bias: Partial	1
13	Summary measures	State the principal summary measures (e.g., risk ratio, difference in means).	649	Yes	2
14	Synthesis of results	Describe the methods of handling data and combining results of studies, if done, including measures of consistency (e.g., I <sup>2</sup> ) for each meta-analysis.	649	Yes: Methodology reported for ES, CI and Q calculations	2
15	Risk of bias across studies	<b>Specify any assessment of risk of bias that may affect the cumulative evidence</b> (e.g., publication bias, selective reporting within studies).	649	No	0
16	Additional analyses	<b>Describe methods of additional analyses (e.g., sensitivity or subgroup analyses, meta-regression), if done, indicating which were pre-specified</b>	649	Analysis of dropouts across treatment types: Partial	1

PRISMA CHECKLIST		Pinquart et al., (2007)		Score	
		Page	Notes		
<b>Results</b>					
17	Study selection	Give numbers of studies screened, assessed for eligibility, and included in the review, with reasons for exclusions at each stage, ideally with a flow diagram.	649	No	0
18	Study characteristics	For each study, present characteristics for which data were extracted (e.g., study size, PICOS, follow-up period) and provide the citations.	649-652	Yes; Appendix	2
19	Risk of bias within studies	Present data on risk of bias of each study and, if available, any outcome level assessment (see item 12).	649-652	No (large number of studies)	0
20	Results of individual studies	For all outcomes considered (benefits or harms), present, for each study: (a) simple summary data for each intervention group (b) effect estimates and confidence intervals, ideally with a forest plot.	649-652	Effect size reported but not CI: Partial	1
21	Synthesis of results	Present results of each meta-analysis done, including confidence intervals and measures of consistency.	650	Yes	2
22	Risk of bias across studies	Present results of any assessment of risk of bias across studies (see Item 15).	651	Analysis of study quality on treatment effects/sample size undertaken. Partial	1
23	Additional analysis	Give results of additional analyses, if done (e.g., sensitivity or subgroup analyses, meta-regression [see Item 16]).	651	yes	2
<b>Discussion</b>					
24	Summary of evidence	Summarize the main findings including the strength of evidence for each main outcome; consider their relevance to key groups (e.g., healthcare providers, users, and policymakers).	652	Limited discussion of the validity/strength of findings but well contextualised: Partial.	1
25	Limitations	Discuss limitations at study and outcome level (e.g., risk of bias), and at review-level (e.g., incomplete retrieval of identified research, reporting bias).	653	Possible review-level bias or limitations not addressed	1
26	Conclusions	Provide a general interpretation of the results in the context of other evidence, and implications for future research.	653	yes	2
<b>Funding</b>					
27	Funding	Describe sources of funding for the systematic review and other support (e.g., supply of data); role of funders for the systematic review.	653	No	0
<b>OVERALL SCORE</b>					<b>30</b>

## PRISMA CHECKLIST

Wilson *et al.*, (2008)

			Page	Notes	Score
<b>Title</b>					
1	Title	Identify the report as a systematic review, meta-analysis, or both.	1	Cochrane Intervention review, sourced from Cochrane database of systematic reviews: so not in title but understood.	2
<b>Abstract</b>					
2	Structured summary	Provide a structured summary including, as applicable: background; objectives; data sources; study eligibility criteria, participants, and interventions; study appraisal and synthesis methods; results; limitations; conclusions and implications of key findings; systematic review registration number.		Yes	2
<b>Introduction</b>					
3	Rationale	Describe the rationale for the review in the context of what is already known.	3	Yes	2
4	Objectives	Provide an explicit statement of questions being addressed with reference to participants, interventions, comparisons, <b>outcomes, and study design (PICOS)</b> .	3	outcomes and study design not specified: Partial	1
<b>Methods</b>					
5	Protocol and registration	Indicate if a review protocol exists, <b>if and where it can be accessed (e.g., Web address), and, if available, provide registration information including registration number.</b>	1	Not included in methods, but elsewhere in paper date of publishing of protocol noted: partial.	1
6	Eligibility criteria	Specify study characteristics (e.g., PICOS, length of follow-up) and report characteristics (e.g., years considered, language, publication status) used as criteria for eligibility, giving rationale.	3-4	Yes	2
7	Information sources	Describe all information sources (e.g., databases with dates of coverage, contact with study authors to identify additional studies) in the search and date last searched.	4	Yes	2
8	Search	Present full electronic search strategy for at least one database, including any limits used, such that it could be repeated.	4	Yes	2
9	Study selection	State the process for selecting studies (i.e., screening, eligibility, included in systematic review, and, if applicable, included in the meta-analysis).	4	Yes	2
10	Data collection process	Describe method of data extraction from reports (e.g., piloted forms, independently, in duplicate) and any processes for obtaining and confirming data from investigators.	4	Yes	2
11	Data items	List and define all variables for which data were sought (e.g., PICOS, funding sources) and any assumptions and simplifications made.	4	Yes	2
12	Risk of bias in individual studies	Describe methods used for assessing risk of bias of individual studies (including specification of whether this was done at the study or outcome level), and how this information is to be used in any data synthesis.	4-5	Yes	2
13	Summary measures	State the principal summary measures (e.g., risk ratio, difference in means).	4	Yes	2
14	Synthesis of results	Describe the methods of handling data and combining results of studies, if done, including <b>measures of consistency (e.g., I<sup>2</sup>) for each meta-analysis.</b>	5	Measures of consistency not reported, but not undertaken	2
15	Risk of bias across studies	Specify any assessment of risk of bias that may affect the cumulative evidence (e.g., publication bias, selective reporting within studies).	5	Yes	2
16	Additional analyses	Describe methods of additional analyses (e.g., sensitivity or subgroup analyses, meta-regression), if done, indicating which were pre-specified	5	Yes	2

PRISMA CHECKLIST		Wilson et al., (2008)		Score	
		Page	Notes		
<b>Results</b>					
17	Study selection	Give numbers of studies screened, assessed for eligibility, and included in the review, with reasons for exclusions at each stage, ideally with a flow diagram.	5	Only post-screening reasons for exclusion noted: but in good detail: Partial.	1
18	Study characteristics	For each study, present characteristics for which data were extracted (e.g., study size, PICOS, follow-up period) and provide the citations.	6-8, 19-26	Yes	2
19	Risk of bias within studies	Present data on risk of bias of each study and, if available, any outcome level assessment (see item 12).	8	Yes	2
20	Results of individual studies	For all outcomes considered (benefits or harms), present, for each study: (a) simple summary data for each intervention group (b) effect estimates and confidence intervals, ideally with a forest plot.	9-19	Detailed outline in text and in table form.	2
21	Synthesis of results	Present results of each meta-analysis done, including confidence intervals and measures of consistency.	31-39	Yes	2
22	Risk of bias across studies	Present results of any assessment of risk of bias across studies (see Item 15).	8-9	Results of funnel plot not reported. Quality Rating Scale mean scores reported: Partial	1
23	Additional analysis	Give results of additional analyses, if done (e.g., sensitivity or subgroup analyses, meta-regression [see Item 16]).	10	Yes	2
<b>Discussion</b>					
24	Summary of evidence	Summarize the main findings including the strength of evidence for each main outcome; consider their relevance to key groups (e.g., healthcare providers, users, and policymakers).	10-11	Yes	2
25	Limitations	Discuss limitations at study and outcome level (e.g., risk of bias), and at review-level (e.g., incomplete retrieval of identified research, reporting bias).	10-11	Yes	2
26	Conclusions	Provide a general interpretation of the results in the context of other evidence, and implications for future research.	11	Yes	2
<b>Funding</b>					
27	Funding	Describe sources of funding for the systematic review and other support (e.g., supply of data); role of funders for the systematic review.	40	Yes	2
<b>OVERALL SCORE</b>					<b>50</b>

## PRISMA CHECKLIST

Peng *et al.*, (2009)

			Page	Notes	Score
<b>Title</b>					
1	Title	Identify the report as a systematic review, meta-analysis, or both.	975	Yes	2
<b>Abstract</b>					
2	Structured summary	Provide a <b>structured summary</b> including, as applicable: background; <b>objectives; data sources; study eligibility criteria, participants, and interventions; study appraisal and synthesis methods; results; limitations; conclusions and implications of key findings; systematic review registration number.</b>	975	Partial	1
<b>Introduction</b>					
3	Rationale	Describe the rationale for the review in the context of what is already known.	976	No rationale in context of what is known.	0
4	Objectives	Provide an explicit statement of questions being addressed with reference to participants, interventions, comparisons, outcomes, and study design (PICOS).	976	Explicit questions not made, and PICOS components not defined.	0
<b>Methods</b>					
3	Rationale	Describe the rationale for the review in the context of what is already known.	976	No rationale in context of what is known.	0
4	Objectives	Provide an explicit statement of questions being addressed with reference to participants, interventions, comparisons, outcomes, and study design (PICOS).	976	Explicit questions not made, and PICOS components not defined.	0
7	Information sources	Describe all information sources (e.g., databases with <b>dates of coverage, contact with study authors to identify additional studies in the search and date last searched.</b>	976	Literature search strategy reported in a separate paper. However that review had separate objectives: (Chang-Quan et al., 2009) and papers for current study, result of this: Partial	1
8	Search	<b>Present full electronic search strategy for at least one database, including any limits used, such that it could be repeated.</b>	976	No	0
9	Study selection	State the process for selecting studies (i.e., screening, eligibility, included in systematic review, and, if applicable, included in the meta-analysis).	976-978	Eligibility criteria includes 'catch all category 'general psychotherapy' which included talking and education about therapy': Partial	1
10	Data collection process	<b>Describe method of data extraction from reports (e.g., piloted forms, independently, in duplicate) and any processes for obtaining and confirming data from investigators.</b>	977	Two reviewers independently assessed: details not clear: Partial.	1
11	Data items	List and define all variables for which data were sought (e.g., PICOS, funding sources) and any assumptions and simplifications made.	976	Study type, participants, interventions defined: Partial	1
12	Risk of bias in individual studies	<b>Describe methods used for assessing risk of bias of individual studies (including specification of whether this was done at the study or outcome level), and how this information is to be used in any data synthesis.</b>	977	No	0
13	Summary measures	<b>State the principal summary measures (e.g., risk ratio, difference in means).</b>	977	No	0
14	Synthesis of results	Describe the methods of handling data and combining results of studies, if done, including measures of consistency (e.g., I <sup>2</sup> ) for each meta-analysis.	977	Very brief. Methodology not clear. No	0
15	Risk of bias across studies	<b>Specify any assessment of risk of bias that may affect the cumulative evidence (e.g., publication bias, selective reporting within studies).</b>	977	No	0
16	Additional analyses	<b>Describe methods of additional analyses (e.g., sensitivity or subgroup analyses, meta-regression), if done, indicating which were pre-specified</b>	977	No	0

PRISMA CHECKLIST		Peng et al., (2009)		Score	
		Page	Notes		
<b>Results</b>					
17	Study selection	Give numbers of studies screened, assessed for eligibility, and included in the review, with reasons for exclusions at each stage, ideally with a flow diagram.	977	Two reasons for exclusion following screening given: Partial	1
18	Study characteristics	For each study, present characteristics for which data were extracted (e.g., study size, PICOS, follow-up period) and provide the citations.	977	No	0
19	Risk of bias within studies	Present data on risk of bias of each study and, if available, any outcome level assessment (see item 12).	977-978	No	0
20	Results of individual studies	For all outcomes considered (benefits or harms), present, for each study: (a) simple summary data for each intervention group (b) effect estimates and confidence intervals, ideally with a forest plot.	978	No	0
21	Synthesis of results	Present results of each meta-analysis done, including confidence intervals and measures of consistency.	978-979	Meta-analysis reported. No measure of consistency: Partial	1
22	Risk of bias across studies	Present results of any assessment of risk of bias across studies (see Item 15).	978-979	No.	0
23	Additional analysis	Give results of additional analyses, if done (e.g., sensitivity or subgroup analyses, meta-regression [see Item 16]).	979	Limited information given: Partial	1
<b>Discussion</b>					
24	Summary of evidence	Summarize the main findings including the strength of evidence for each main outcome; consider their relevance to key groups (e.g., healthcare providers, users, and policy makers).	979-980	Summary not structured, without critical analysis: Partial	1
25	Limitations	Discuss limitations at study and outcome level (e.g., risk of bias), and at review-level (e.g., incomplete retrieval of identified research, reporting bias).	980	Impact of publication bias not defined: Bias related to outcome measure depression mentioned. Small sample size noted: Partial	1
26	Conclusions	Provide a general interpretation of the results in the context of other evidence, and implications for future research.	981	Results not interpreted, simply restated: No.	0
<b>Funding</b>					
27	Funding	Describe sources of funding for the systematic review and other support (e.g., supply of data); role of funders for the systematic review.	981	Reported no conflict of interests: but details of funding not outlined: No	0
<b>OVERALL SCORE</b>					<b>12</b>



PRISMA CHECKLIST

Samad *et al.*, (2011)

			Page	Notes	Score
<b>Title</b>					
1	Title	Identify the report as a systematic review, meta-analysis, or both.	1211	Yes	2
<b>Abstract</b>					
2	Structured summary	Provide a structured summary including, as applicable: <b>background</b> ; objectives; data sources; study eligibility criteria, participants, and interventions; study appraisal and synthesis methods; results; <b>limitations</b> ; conclusions and implications of key findings; systematic review registration number.	1211	structured summary including key details.	2
<b>Introduction</b>					
3	Rationale	Describe the rationale for the review in the context of what is already known.	1212	Yes	2
4	Objectives	Provide an explicit statement of questions being addressed with reference to participants, interventions, comparisons, outcomes, and study design (PICOS).	1212	Yes	2
<b>Methods</b>					
5	Protocol and registration	<b>Indicate if a review protocol exists, if and where it can be accessed (e.g., Web address), and, if available, provide registration information including registration number.</b>	1212	No	0
6	Eligibility criteria	Specify study characteristics (e.g., PICOS, length of follow-up) and report characteristics (e.g., years considered, language, publication status) used as criteria for eligibility, giving rationale.	1212-1213	Yes	2
7	Information sources	Describe all information sources (e.g., databases with dates of coverage, <b>contact with study authors to identify additional studies</b> ) in the search and <b>date last searched</b> .	1212-1213	Yes, although date of last search not given: Partial	1
8	Search	<b>Present full electronic search strategy for at least one database, including any limits used, such that it could be repeated.</b>		No	0
9	Study selection	State the process for selecting studies (i.e., screening, eligibility, included in systematic review, and, if applicable, included in the meta-analysis).	1212-1213	yes	2
10	Data collection process	Describe method of data extraction from reports (e.g., piloted forms, independently, in duplicate) and any processes for obtaining and confirming data from investigators.	1214	Yes	2
11	Data items	List and define all variables for which data were sought (e.g., PICOS, funding sources) and any assumptions and simplifications made.	1213	Yes	2
12	Risk of bias in individual studies	Describe methods used for assessing risk of bias of individual studies (including specification of whether this was done at the study or outcome level), and how this information is to be used in any data synthesis.	1213	Yes	2
13	Summary measures	State the principal summary measures (e.g., risk ratio, difference in means).	1213	Yes	2
14	Synthesis of results	Describe the methods of handling data and combining results of studies, if done, including measures of consistency (e.g., I <sup>2</sup> ) for each meta-analysis.	1214	Yes	2
15	Risk of bias across studies	Specify any assessment of risk of bias that may affect the cumulative evidence (e.g., publication bias, selective reporting within studies).	1214	Yes: risk of bias of individual studies is reported and it is noted that the implications for results is examined narratively.	2
16	Additional analyses	<b>Describe methods of additional analyses (e.g., sensitivity or subgroup analyses, meta-regression), if done, indicating which were pre-specified</b>	1214	Sub-group analyses not noted.	0



PRISMA CHECKLIST		Samad et al., (2011)		Score	
		Page	Notes		
<b>Results</b>					
17	Study selection	Give numbers of studies screened, assessed for eligibility, and included in the review, with reasons for exclusions at each stage, ideally with a flow diagram.	1214-1215	Yes	2
18	Study characteristics	For each study, present characteristics for which data were extracted (e.g., study size, PICOS, follow-up period) and provide the citations.	1216	Yes	2
19	Risk of bias within studies	Present data on risk of bias of each study and, if available, any outcome level assessment (see item 12).	1214-1215	Yes	2
20	Results of individual studies	For all outcomes considered (benefits or harms), present, for each study: (a) simple summary data for each intervention group (b) effect estimates and confidence intervals, ideally with a forest plot.	1217	Yes	2
21	Synthesis of results	Present results of each meta-analysis done, including confidence intervals and measures of consistency.	1217-1218	Yes	2
22	Risk of bias across studies	Present results of any assessment of risk of bias across studies (see Item 15).	1216	Data for within studies considered here (only 4 studies included) : partial	1
23	Additional analysis	Give results of additional analyses, if done (e.g., sensitivity or subgroup analyses, meta-regression [see Item 16]).		Not undertaken	0
<b>Discussion</b>					
24	Summary of evidence	Summarize the main findings including the strength of evidence for each main outcome; consider their relevance to key groups (e.g., healthcare providers, users, and policymakers).	1219	Yes	2
25	Limitations	Discuss limitations at study and outcome level (e.g., risk of bias), and at review-level (e.g., incomplete retrieval of identified research, reporting bias).	1218	Yes	2
26	Conclusions	Provide a general interpretation of the results in the context of other evidence, and implications for future research.	1219	Yes	2
<b>Funding</b>					
27	Funding	Describe sources of funding for the systematic review and other support (e.g., supply of data); role of funders for the systematic review.	1219	Yes	2
<b>OVERALL SCORE</b>					44

## PRISMA CHECKLIST

Krishna *et al.*, (2011)

			Page	Notes	Score
<b>Title</b>					
1	Title	Identify the report as a systematic review, meta-analysis, or both.	331	Yes	2
<b>Abstract</b>					
2	Structured summary	Provide a structured summary including, as applicable: <b>background</b> ; objectives; data sources; study <b>eligibility criteria</b> , participants, and interventions; <b>study appraisal and synthesis methods</b> ; results; <b>limitations</b> ; conclusions and <b>implications of key findings</b> ; systematic review registration number.	331	Partial	1
<b>Introduction</b>					
3	Rationale	Describe the rationale for the review in the context of what is already known.	331-332	Yes	2
4	Objectives	Provide an explicit statement of questions being addressed with reference to <b>participants, interventions</b> , comparisons, outcomes, and <b>study design (PICOS)</b> .	332	Partial	1
<b>Methods</b>					
5	Protocol and registration	<b>Indicate if a review protocol exists, if and where it can be accessed (e.g., Web address), and, if available, provide registration information including registration number.</b>	332-333	No	0
6	Eligibility criteria	Specify study characteristics (e.g., PICOS, length of follow-up) and report characteristics (e.g., years considered, language, publication status) used as criteria for eligibility, giving rationale.	332-333	Yes	2
7	Information sources	Describe all information sources (e.g., databases with dates of coverage, contact with study authors to identify additional studies) in the search and date last searched.	332	Yes	2
8	Search	<b>Present full electronic search strategy for at least one database, including any limits used, such that it could be repeated.</b>		No	0
9	Study selection	<b>State the process for selecting studies (i.e., screening, eligibility, included in systematic review, and, if applicable, included in the meta-analysis).</b>	332-333	Process not described systematically; outlining at what stage, for example, studies were excluded: Partial	1
10	Data collection process	Describe method of data extraction from reports (e.g., <b>piloted forms, independently, in duplicate</b> ) and any processes for obtaining and confirming data from investigators.	333	Data extraction tool mentioned, but methods not detailed: Partial	1
11	Data items	<b>List and define all variables for which data were sought</b> (e.g., PICOS, funding sources) and any assumptions and simplifications made.	333	Variables not listed and defined in methods: No	0
12	Risk of bias in individual studies	Describe methods used for assessing risk of bias of individual studies (including specification of whether this was done at the study or outcome level), and how this information is to be used in any data synthesis.	333	QRS and Higgins and Green (2006) used, and drop-out calculated: plans to use quality measure in sub-group analysis noted: Yes.	2
13	Summary measures	State the principal summary measures (e.g., risk ratio, difference in means).	333	mean differences / odds ratio: Yes	2
14	Synthesis of results	Describe the methods of handling data and combining results of studies, if done, <b>including measures of consistency (e.g., I<sup>2</sup>) for each meta-analysis.</b>	333	Methods not detailed. t <sup>2</sup> test for heterogeneity used: Partial.	1
15	Risk of bias across studies	Specify any assessment of risk of bias that may affect the cumulative evidence (e.g., publication bias, selective reporting within studies).	333	Yes	2
16	Additional analyses	Describe methods of additional analyses (e.g., <b>sensitivity or subgroup analyses</b> , meta-regression), <b>if done, indicating which were pre-specified</b>	333	Methods for sensitivity/sub-group analyses not given, but noted: Partial.	1

PRISMA CHECKLIST			Krishna et al., (2011)		
			Page	Notes	Score
<b>Results</b>					
17	Study selection	Give numbers of studies screened, assessed for eligibility, and included in the review, with reasons for exclusions at each stage, ideally with a flow diagram.	333	Yes	2
18	Study characteristics	For each study, present characteristics for which data were extracted (e.g., study size, PICOS, follow-up period) and provide the citations.	333-336	Yes	2
19	Risk of bias within studies	Present data on risk of bias of each study and, if available, any outcome level assessment (see item 12).	333-336	Yes	2
20	Results of individual studies	For all outcomes considered (benefits or harms), present, for each study: (a) simple summary data for each intervention group (b) effect estimates and confidence intervals, ideally with a forest plot.	337	Yes: number of studies noted to be too small for funnel plot and sensitivity analysis.	2
21	Synthesis of results	Present results of each meta-analysis done, including confidence intervals and <b>measures of consistency</b> .	337-338	Mean diff, ES and CI, No measure of consistency: Partial.	1
22	Risk of bias across studies	Present results of any assessment of risk of <b>bias across</b> studies (see Item 15).	337-338	Partial but not detailed	1
23	Additional analysis	<b>Give results of additional analyses, if done (e.g., sensitivity or subgroup analyses, meta-regression [see Item 16]).</b>		No	0
<b>Discussion</b>					
24	Summary of evidence	Summarize the main findings including the strength of evidence for each main outcome; consider their relevance to key groups (e.g., healthcare providers, users, and policy makers).	338-339	Yes	2
25	Limitations	Discuss limitations at study and outcome level (e.g., risk of bias), and at <b>review-level</b> (e.g., incomplete retrieval of identified research, reporting bias).	338-339	Study level limitations well described, but review-level not; Partial	1
26	Conclusions	Provide a general interpretation of the results in the context of other evidence, and implications for future research.	338-339	Yes	2
<b>Funding</b>					
27	Funding	Describe sources of funding for the systematic review and other support (e.g., supply of data); role of funders for the systematic review.	339	Yes	2
<b>OVERALL SCORE</b>					<b>37</b>

## Appendix 7: Excluded studies (Systematic Review).

Authors	Year	Title	Journal	Vol.	Pages	Reason for Exclusion
Cuijpers, P., Andersson, G., Donker, T., van Straten, A.	2011	Psychological treatment of depression: Results of a series of meta-analyses	Nordic Journal of Psychiatry	65(6)	354-364	Although one sub-group analysis of late-life depression is included this is minimal, and only examines general efficacy as compared with younger adults.
Pinquart, M., Duberstein, P.R., Lyness, J.M.	2006	Effects of pharmacotherapy and psychotherapy in late-life depression	Brown University Psychopharmacology Update	17 (12):	6-7	Summary of Pinquart M, Duberstein PR, Lyness JM (2006)
Cole M.G., Elie L.M., McCusker, J., Bellavance, F., Mansour, A.	2000	Feasibility and effectiveness of treatments for depression in elderly medical inpatients: a systematic review.	International Psycho-geriatrics	12 (4)	453-61	Systematic summary of medication, rather than psychosocial interventions: also lacking meta-analytic methods to examine treatment specificity when concomitant psychotherapeutic treatment included.
Freudenstein, U., Jagger. C., Arthur, A., Donner-Banzhoff, N.	2001	Treatments for late life depression in primary care: a systematic review.	Family Practice	18(3)	321-7	Systematic summary without meta-analytic methods to examine treatment specificity.
Lin Y.C., Dai, Y.T., Hwang, S.L.,	2003	The effect of reminiscence on the elderly population: a systematic review	Public Health Nursing	20 (4)	297-306	Descriptive review without meta-analytic methods to examine treatment specificity.
Woods, B	2004	Review: reminiscence and life review are effective therapies for depression in the elderly.	Evidence Based Mental Health	7 (3)		Summary article of: Bohlmeijer et al., (2003)
Frazer, C.J., Christensen, H., Griffiths, K.M.,	2005	Effectiveness of treatments for depression in older people	The Medical Journal Of Australia	182 (12)	627-32	Only summary of evidence: no synthesis or further analysis with meta-analytic methods.
Hill. A., Brettle, A.,	2005	The effectiveness of counselling with older people: results of a systematic review	Counselling & Psychotherapy Research	5(4)	265-72	Summary of evidence. no synthesis or further analysis with meta-analytic methods
Hill. A., Brettle, A.,	2006	Counselling older people: what can we learn from research evidence?	Journal of Social Work Practice	20(3)	281-97	Summary of evidence. no synthesis or further analysis with meta-analytic methods
Price, L.	2006	Treating late-life depression: pharmacotherapy or psychotherapy?	Brown University Geriatric Psychopharmacology Update,	10(12)	3-4	Summary article of Treatments for later-life depressive conditions: a meta-analytic comparison of pharmacotherapy and psychotherapy.
Cole, M.G.	2008	Brief interventions to prevent depression in older subjects: A systematic review of feasibility and effectiveness	The American Journal of Geriatric Psychiatry	16(6)	435-443	Examining preventative approaches: so not currently depressed. Also Age group 40+ also no meta-analytic methods to examine subgroup treatment specificity.
Adamek, M.E., Slater, G.Y.	2008	Depression and anxiety.	Journal of Gerontological Social Work,	50 (S1)	153-89	No synthesis or further analysis with meta-analytic methods

Authors	Year	Title	Journal	Vol.	Pages	Reason for Exclusion
Payne, T.P., Marcus, D.K.	2008	The efficacy of Group Psychotherapy for Older Adult Clients: A meta-analysis	Group Dynamics, Theory, Research and Practice	12(4)	268-278	Not looking at Depression specifically, with no mediator analysis that could be usefully extracted with regard to treatments for depression.
Forsman, A.K., Schierenbeck, I., Wahlbeck, K.,	2011	Psychosocial Interventions for the Prevention of Depression in Older Adults: Systematic Review and Meta-Analysis.	Journal of Aging & Health	23(3)	387-416	Prevention rather than treatment.
Forsman, A.K., Nordmyr, J., Wahlbeck, K.,	2011	Psychosocial interventions for the promotion of mental health and the prevention of depression among older adults.	Health Promotion International	26(1)	85-107	Prevention rather than treatment.
Dai, B. Li, J., Cuijpers, P.	2011	Psychological treatment of depressive symptoms in Chinese elderly inpatients with significant medical co-morbidity: a meta-analysis.	BMC Psychiatry	11(1)	92	Physical co-morbidity(also poor quality of included trials)
Cuijpers, P., van Straten, A., Warmerdam, L., Andersson, G.		Psychotherapy versus the combination of psychotherapy and pharmacotherapy in the treatment of depression: A meta-analysis.	Depression and Anxiety	26(3)	279-288	Not specifically late life depression
Hsieh H; Wang J;	2003	Effect of reminiscence therapy on depression in older adults: a systematic review	International Journal of Nursing Studies	40 (4)	335-45	Systematic review without meta-analytic analysis
Kiosses DN; Leon AC; Areán PA	2011	Psychosocial Interventions for Late-life Major Depression: Evidence-Based Treatments, Predictors of Treatment Outcomes, and Moderators of Treatment Effects.	Psychiatric Clinics of North America	43(2)	377-401	No meta-analytic methods
Koder, D., Brodaty, H., Anstey, K.	1996	Cognitive therapy for depression in the elderly	International journal of geriatric psychiatry	11(2)	97-107	No, effect sizes calculated for some studies, but not a meta-analysis

## Appendix 8: Calculation of Kappa statistic (Systematic Review).

**Calculation of Kappa statistic based on the PRISMA ratings, provided by two independent observers, of seven of the included studies selected at random.**

		Observer 1			
		0	1	2	Total
Observer 2	0	37	2	0	<b>39</b>
	1	5	45	5	<b>55</b>
	2	0	9	86	<b>95</b>
	Total	<b>42</b>	<b>56</b>	<b>91</b>	<b>189</b>

Calculations, following Viera & Garrett (2005)

Observed agreement

$P_o$  0.88888889

Number of observed agreements: 168 (88.89% of the observations)

Expected agreement

$P_e$  0.37409367

Number of agreements expected by chance: 70.7 (37.41% of the observations)

Kappa

$K = (P_o - P_e) / (1 - P_e) = 0.822479649$

Calculations corroborated at:

<http://graphpad.com/quickcalcs/kappa1.cfm?K=3>

Number of observed agreements: 168 ( 88.89% of the observations)

Number of agreements expected by chance: 70.7 ( 37.41% of the observations)

Kappa= 0.822

SE of kappa = 0.036

95% confidence interval: From 0.751 to 0.894

The strength of agreement is considered to be 'very good'.

**Reference**

Viera, A.J., & Garrett, J.M. (2005). Understanding interobserver agreement: the kappa statistic. Family medicine, 37(5), 360-3.



## Appendix 9: Regression Analyses (Systematic Review).

## Reporting Quality / Year of Publication

Call:

```
lm(formula = Rep_Qual ~ Year)
```

Residuals:

Min	1Q	Median	3Q	Max
-22.282	-2.462	1.166	2.274	12.614

Coefficients:

	Estimate	Std. Error	t value	Pr(> t )
(Intercept)	-1765.4521	732.0817	-2.412	0.0328 *
Year	0.8953	0.3654	2.450	0.0306 *

---

Signif. codes: 0 '\*\*\*' 0.001 '\*\*' 0.01 '\*' 0.05 '.' 0.1 ' ' 1

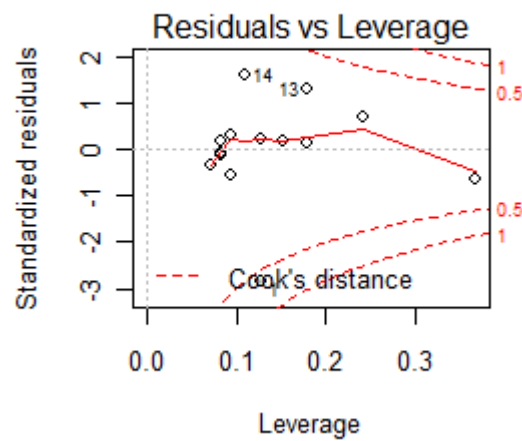
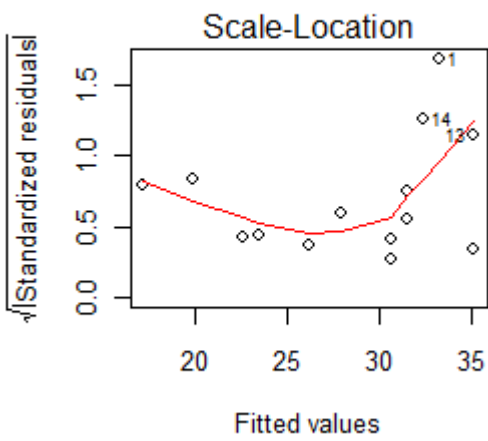
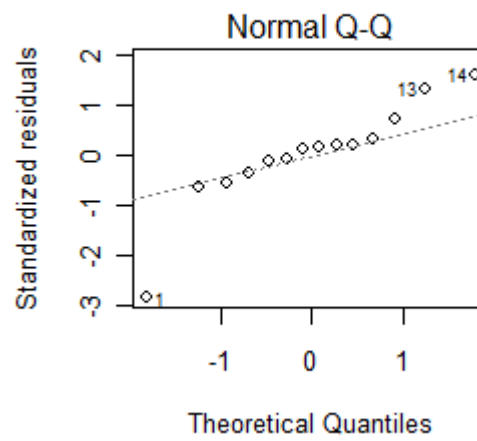
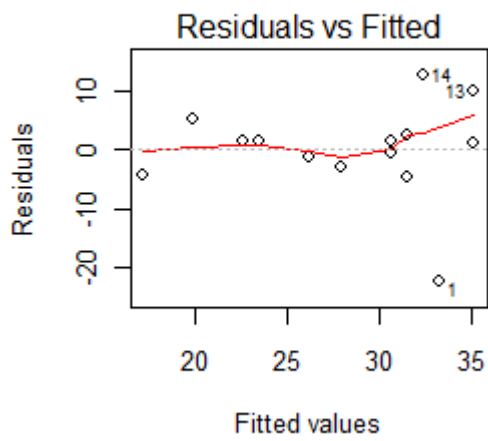
Residual standard error: 8.376 on 12 degrees of freedom

Multiple R-squared: 0.3335, Adjusted R-squared: 0.2779

F-statistic: 6.004 on 1 and 12 DF, p-value: 0.03058

$Y = mX + c$

Rep\_Qual = 0.8953 x Year + -1765.4521



Model validation plots

---

## Number of Analyses / Year of Publication

Call:

```
lm(formula = Analyses ~ Year)
```

Residuals:

Min	1Q	Median	3Q	Max
-3.7678	-1.9018	0.1199	0.8661	6.2322

Coefficients:

	Estimate	Std. Error	t value	Pr(> t )
(Intercept)	710.323	240.507	2.953	0.0121 *
Year	-0.352	0.120	-2.933	0.0125 *

---

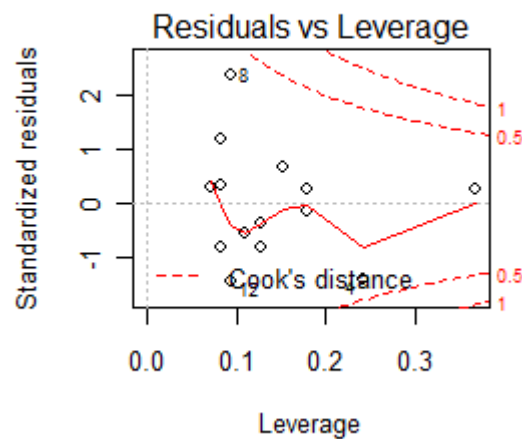
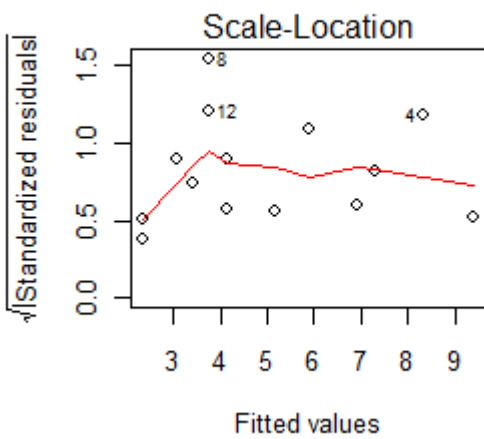
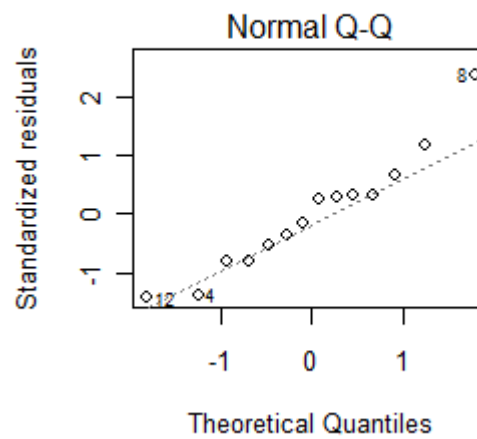
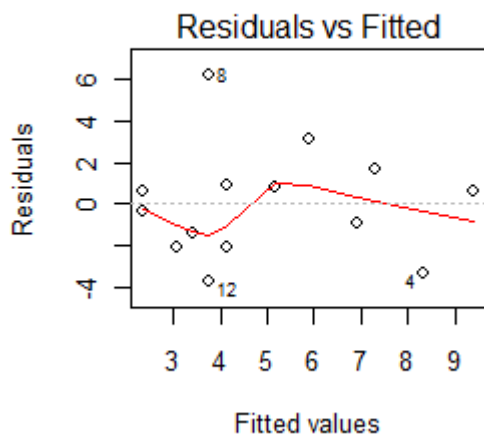
Signif. codes: 0 '\*\*\*' 0.001 '\*\*' 0.01 '\*' 0.05 '.' 0.1 ' ' 1

Residual standard error: 2.752 on 12 degrees of freedom

Multiple R-squared: 0.4175, Adjusted R-squared: 0.3689

F-statistic: 8.601 on 1 and 12 DF, p-value: 0.01254

Analyses = -0.352 Year + 710.323



Model validation plots

## Number of analyses / Reporting Quality

Call:

```
lm(formula = Analyses ~ Rep_Qual)
```

Residuals:

Min	1Q	Median	3Q	Max
-6.6244	-1.5102	0.4924	2.1462	4.7948

Coefficients:

	Estimate	Std. Error	t value	Pr(> t )
(Intercept)	9.28764	2.73863	3.391	0.00535 **
Rep_Qual	-0.15120	0.09158	-1.651	0.12462

---

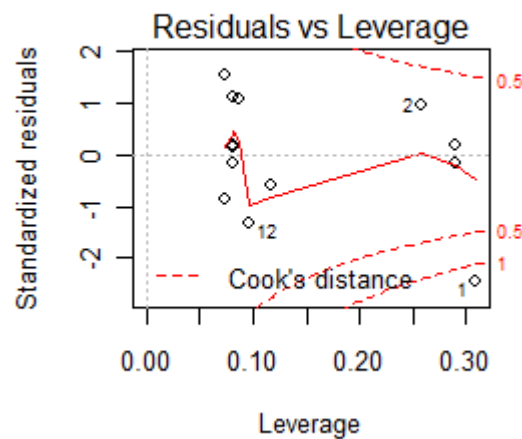
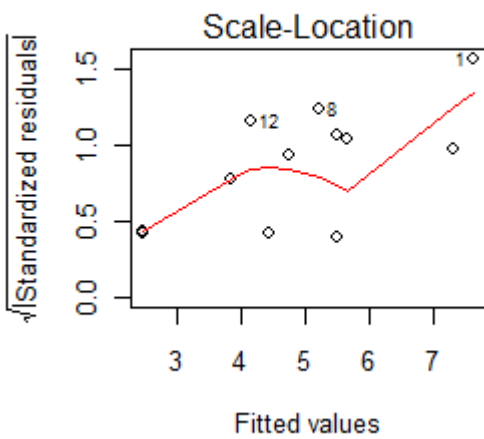
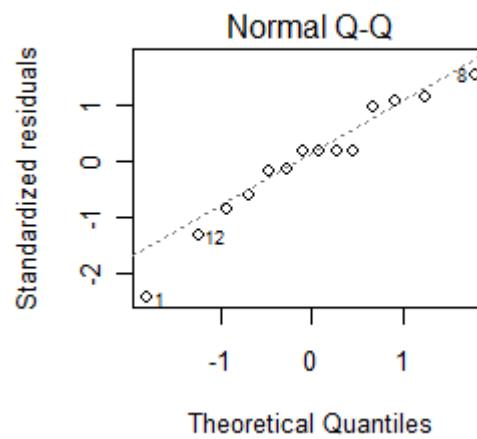
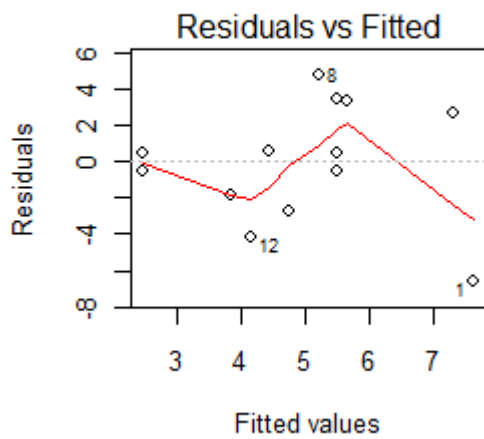
Signif. codes: 0 '\*\*\*' 0.001 '\*\*' 0.01 '\*' 0.05 '.' 0.1 ' ' 1

Residual standard error: 3.255 on 12 degrees of freedom

Multiple R-squared: 0.1851, Adjusted R-squared: 0.1172

F-statistic: 2.726 on 1 and 12 DF, p-value: 0.1246

Analyses = -0.15120 Rep\_Qual + 9.28764



Model validation plots

Cook's value > 1 for Peng 2009

= outlier

## Number of analyses / Reporting Quality without Peng 2009 (outlier)

Call:

```
lm(formula = Analyses ~ Rep_Qual)
```

Residuals:

Min	1Q	Median	3Q	Max
-4.0882	-1.5220	-0.0333	2.0260	3.9301

Coefficients:

	Estimate	Std. Error	t value	Pr(> t )	
(Intercept)	13.71370	2.42242	5.661	0.000146	***
Rep_Qual	-0.28310	0.07844	-3.609	0.004102	**

---

Signif. codes: 0 '\*\*\*' 0.001 '\*\*' 0.01 '\*' 0.05 '.' 0.1 ' ' 1

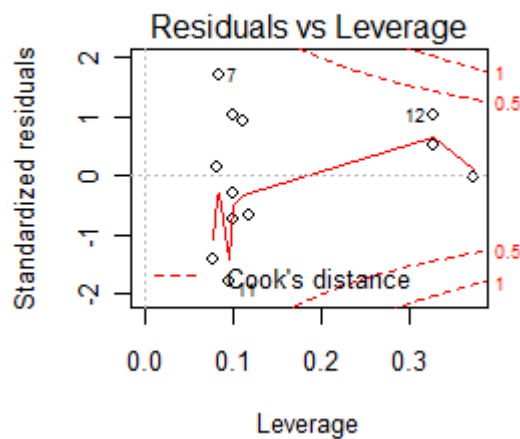
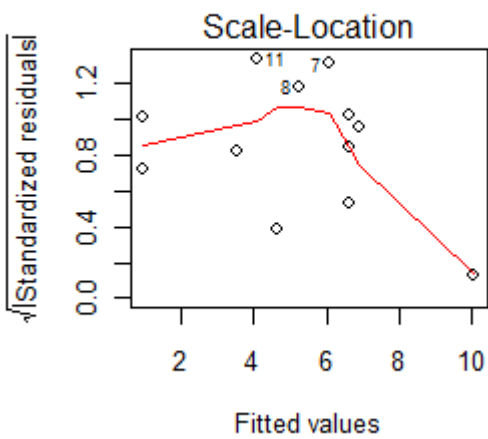
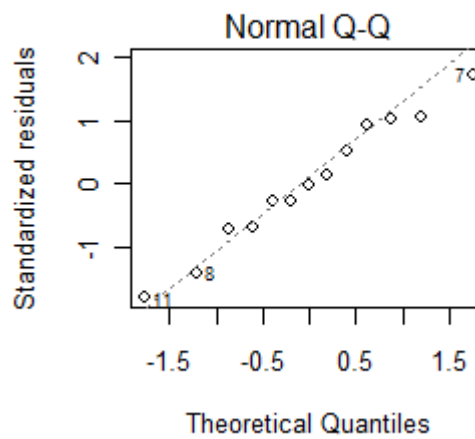
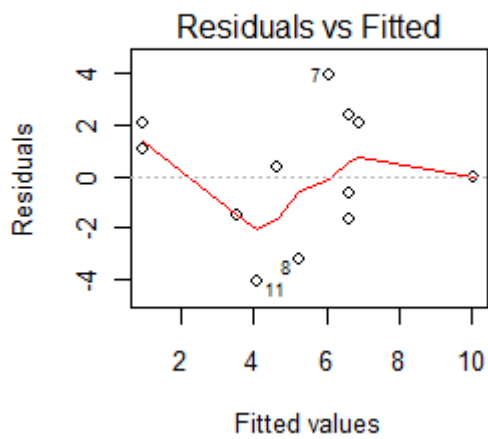
Residual standard error: 2.403 on 11 degrees of freedom

Multiple R-squared: 0.5422, Adjusted R-squared: 0.5006

F-statistic: 13.03 on 1 and 11 DF, p-value: 0.0041027

Analyses = -0.28310 Rep\_Qual + 13.71370





Model validation plots

Appendix 10: Email correspondence with authors of selected studies (Meta-Analysis).

**Initial E-mail sent to study authors where no further data was required:**

Hello,

I am undertaking a meta-analysis examining CBT for depression with older adults with co-morbid physical illness. This is in part-fulfilment of a Doctorate in Clinical Psychology at Edinburgh University.

Your paper: [.....] meets the inclusion criteria.

I was wondering if you were aware of any unpublished/current RCT trials examining CBT treatment for depression in older adults with a physical illness, which I may not have been able to find via comprehensive literature searches of available databases. My criteria are: mean age ≥55yrs; CBT treatment for mood disorder; co-morbid physical illness; validated outcome measure for depressive symptoms.

If you are aware of any relevant studies it would be fantastic if you could get in contact.

Many thanks,

David Huxtable

David Huxtable Specialist Psychological Practitioner Older Adult Psychology Dept Royal Cornhill Hospital, Aberdeen, AB25 2ZH (01224) 557 497

**Responses:**

Authors and Year	Contacted	Reply
Gellis, Z. D., McGinty, J., Tierney, L., Jordan, C., Burton, J., & Misener, E. (2007)	Y	N
Dao, T. K., Youssef, N. a, Armsworth, M., Wear, E., Papatopoulos, K. N., & Gopaldas, R. (2011).	Y	N
Freedland, K. E., Skala, J. A., Carney, R. M., Rubin, E. H., Lustman, P. J., Da, V. G. (2009).	Y	Y
Hynninen, M. J., Bjerke, N., Pallesen, S., Bakke, P. S., & Nordhus, I. H. (2010)	Y	Y
Koertge, J., Janszky, I., Sundin, O., Blom, M., Georgiades, a, László, K. D., Alinaghizadeh, H., et al. (2008).	Y	N
Kunik, M. E., Veazey, C., Cully, J. A., Soucek, J., Graham, D. P., Hopko, D., Carter, R., et al. (2008).	Y	Y
Lincoln, N.B. Flannaghan, T. (2003).	Y	Y
Lustman, P. J., Griffith, L. S., Freedland, K. E., Kissel, S. S., & Clouse, R. E. (1998).	Y	N
Moorey, S., Cort, E., Kapari, M., Monroe, B., Hansford, P., Mannix, K., Henderson, M., et al. (2009)	Y	N
Dobkin, R. D., Menza, M., Allen, L. A., Gara, M. A., Mark, M. H., Tiu, J., & Bienfait, K. L. (2011)	Y	N
Pibernik-Okanovic, M., Begic, D., Ajdukovic, D., Andrijasevic, N., & Metelko, Z. (2009).	Y	Y
Teri, L., Logsdon, R. G., Uomoto, J., & McCurry, S. M. (1997).	Y	N

---

**Responses to above e-mail:**

1.

Kunik, Mark Edwin [mkunik@bcm.edu]

Sent: 01 May 2012 17:30

To: Huxtable David (NHS GRAMPIAN)

Subject: RE: research related to: COPD education and cognitive behavioral therapy group treatment for clinically significant symptoms of depression and anxiety in COPD patients: a randomized controlled trial.

My colleague, Jeff Cully, PhD is doing a study that would be of interest to you.

I think you can find some of his pilot work on pubmed, but he is ongoing funded study.

jcully@bcm.edu

Mark E. Kunik, M.D., M.P.H.

Associate Director Houston VA Health Services Research and Development Center of Excellence Associate Director for Research Training, South Central MIRECC Professor, Menninger Department of Psychiatry and Behavioral Sciences <http://www.houston.hsrd.research.va.gov/health-services/kunik.htm>

2.

From: Kia Minna Johanna Hynninen [Minna.Hynninen@psykp.uib.no]

Sent: 04 May 2012 10:09

To: Huxtable David (NHS GRAMPIAN)

Subject: RE: research related to:A randomized controlled trial of cognitive behavioral therapy for anxiety and depression in COPD

Hello David,

We do not have any unpublished RCT trials going on, and I am not aware of other such studies.

Good luck with your work!

Minna

3.

From: David C Mohr [d-mohr@northwestern.edu]

Sent: 02 May 2012 13:44

To: Huxtable David (NHS GRAMPIAN)

Subject: RE: query re: The Effect of Telephone-Administered Cognitive-Behavioral Therapy on Quality of Life among Patients with Multiple Sclerosis

Attachments: 2005 Arch Gen Psychiatr.pdf

Hi,

That was a secondary analysis. Attached is the main paper.

---

D

David C. Mohr, Ph.D.  
www.cbits.northwestern.edu

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4.

From: Freedland, Ken [freedlak@bmc.wustl.edu]  
Sent: 02 May 2012 20:35  
To: Huxtable David (NHS GRAMPIAN)  
Subject: RE: research related to: Treatment of Depression After Coronary Artery Bypass Surgery

Hi David,

Our ongoing randomized trial of CBT for depression in patients with heart failure fits your criteria. We're still enrolling patients and haven't done any analyses yet. You can find more info about it at [clinicaltrials.gov](http://clinicaltrials.gov).

You might also want to check with David Mohr at Northwestern University in Chicago. d-mohr@northwestern.edu

Good luck with your meta-analysis.

Ken

Kenneth E. Freedland, PhD  
Professor of Psychiatry  
Washington University School of Medicine 4320 Forest Park Ave., Suite 301 St. Louis, Missouri 63108 USA  
314-286-1311 (phone)  
314-286-1301 (fax)

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**Correspondence requesting further data:**

5.

Huxtable David (NHS GRAMPIAN)  
Sent: 08 May 2012 20:33  
To: pibernik@idb.hr  
Subject: request for data: 'Psychoeducation versus treatment as usual in diabetic patients with subthreshold depression: preliminary results of a randomized controlled trial'

Hello,

I am undertaking a meta-analysis examining cognitive-behavioural treatments for depression in older adults with co-morbid physical illness. This is in part-fulfilment of a Doctorate in Clinical Psychology at Edinburgh University.

I was wondering if you might be able to assist me. Your paper: 'Psychoeducation versus treatment as usual in diabetic patients with subthreshold depression: preliminary results of a randomized controlled trial' meets the inclusion criteria for my study. I am keen to include the paper; however, I require some additional information.

In table 2 you report changes in CES-D scores in this format: 26 (22–30) to 18 (12.5–28.5). I understand this to be the Median (25–75)

To include the data my analyses I require mean and standard deviation data for baseline and outcome scores on the CES-D for both intervention and control groups.

If you were able to give me these data, it would be fantastic, and allow me to include your paper in my study.

Any help would be much appreciated.

David Huxtable Specialist Psychological Practitioner Older Adult Psychology Dept Royal Cornhill Hospital,  
Aberdeen, AB25 2ZH  
(01224) 557 497

-reply-

From: Nadina Lincoln [Nadina.Lincoln@nottingham.ac.uk]  
Sent: 06 May 2012 07:53  
To: Huxtable David (NHS GRAMPIAN)  
Subject: RE: request for data re: Cognitive Behavioral Psychotherapy for Depression Following Stroke

Hi

I am happy to provide the data. However, I am about to go away for 3 weeks, so it will not be until I get back on 26th May. Sorry about the delay. I hope that it will not be too late for you.

Regards

Nadina

From: Huxtable David (NHS GRAMPIAN)  
Sent: 06 May 2012 15:18  
To: Nadina Lincoln  
Subject: RE: request for data re: Cognitive Behavioral Psychotherapy for Depression Following Stroke

-reply-

Hi,

It would be great if you could supply the data on your return.

Many thanks,

David

David Huxtable Specialist Psychological Practitioner Older Adult Psychology Dept Royal Cornhill Hospital,  
Aberdeen, AB25 2ZH  
(01224) 557 497

---

From: Nadina Lincoln [Nadina.Lincoln@nottingham.ac.uk]  
Sent: 23 May 2012 08:21  
To: Huxtable David (NHS GRAMPIAN)  
Subject: RE: request for data re: Cognitive Behavioral Psychotherapy for Depression Following Stroke

Attachments: Frequencies 3m FU.doc; Frequencies 6 months.doc

Attached

Nadina

Nadina Lincoln  
Professor of Clinical Psychology  
University of Nottingham  
International House  
Jubilee Campus  
Nottingham NG8 1BB

0115 9515315 ((Monday, Thursday, Friday))

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6.

From: Huxtable David (NHS GRAMPIAN)  
Sent: 08 May 2012 20:33

To: pibernik@idb.hr  
Subject: request for data: 'Psychoeducation versus treatment as usual in diabetic patients with subthreshold depression: preliminary results of a randomized controlled trial'

Hello,

I am undertaking a meta-analysis examining cognitive-behavioural treatments for depression in older adults with co-morbid physical illness. This is in part-fulfillment of a Doctorate in Clinical Psychology at Edinburgh University.

I was wondering if you might be able to assist me. Your paper: 'Psycho education versus treatment as usual in diabetic patients with sub threshold depression: preliminary results of a randomized controlled trial' meets the inclusion criteria for my study. I am keen to include the paper; however, I require some additional information.

In table 2 you report changes in CES-D scores in this format: 26 (22–30) to 18 (12.5–28.5). I understand this to be the Median (25–75)

To include the data my analyses I require mean and standard deviation data for baseline and outcome scores on the CES-D for both intervention and control groups.

If you were able to give me these data, it would be fantastic, and allow me to include your paper in my study.

Any help would be much appreciated.

David Huxtable Specialist Psychological Practitioner Older Adult Psychology Dept Royal Cornhill Hospital,  
Aberdeen, AB25 2ZH  
(01224) 557 497

From: Mirjana Pibernik-Okanovic [pibernik@idb.hr]  
Sent: 09 May 2012 17:52  
To: Huxtable David (NHS GRAMPIAN)  
Subject: Re: request for data: 'Psychoeducation versus treatment as usual in diabetic patients with subthreshold depression: preliminary results of a randomized controlled trial'

Dear Dr.Huxtable,

I have added the information about means and standard deviations in the CESD scores obtained at baseline, 6 month- and 12-month follow up.

Intervention arm:

baseline  $M=27.3\pm 7.8$   
6 months  $M=20.1\pm 12.7$   
12 months  $M=17.5\pm 9.7$

Control arm:

baseline  $M=25.4\pm 10.4$   
6 months  $M=20.7\pm 10.5$   
12 months  $M=20.3\pm 12.3$

You must be aware of the asymmetric distribution of the obtained data.

Best regards,  
Mirjana Pibernik-Okanovic

From: Huxtable David (NHS GRAMPIAN)  
Sent: 13 May 2012 22:41  
To: Mirjana Pibernik-Okanovic  
Subject: A couple more things...

Hi,  
That is great,

Can I ask a couple more things: were the intervention and control groups both  $N=25$ , and do you have data with regard to any drop-out? If there were drop-outs are the means and SD given below based on intention to treat analyses, or based on completer samples?

Also with regard to the skew of the obtained data, are you referring to the differences in education, diet and physical functioning?

It would be fantastic if you could give me this extra information as I would need this to include in the analysis.

Many thanks

David

David Huxtable Specialist Psychological Practitioner Older Adult Psychology Dept Royal Cornhill Hospital,  
Aberdeen, AB25 2ZH  
(01224) 557 497



-No further reply-

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7.

Huxtable David (NHS GRAMPIAN)  
Sent: 08 May 2012 20:19  
To: lteri@u.washington.edu  
Subject: request for data: 'Behavioral treatment of depression in dementia patients: a controlled clinical trial'

Hello,

I am undertaking a meta-analysis examining cognitive-behavioural treatments for depression in older adults with co-morbid physical illness. This is in part-fulfillment of a Doctorate in Clinical Psychology at Edinburgh University.

I was wondering if you might be able to assist me. Your paper: 'Behavioral treatment of depression in dementia patients: a controlled clinical trial' meets the inclusion criteria for my study. I am keen to include the paper, however, I just need to clarify the data and perhaps require some additional information.

In table 1 of your study, baseline scores are reported in this format: HDRS 16.3 ± 5.3 I would like to confirm that the initial number is the mean score and the second number, the standard deviation. It would be great if you could confirm that this is the case. If so, I do not need to request this data for baseline measures.

Secondly, in table 2 you have reported 'Changes in Outcome Measure Scores from Pre- to Post-treatment'. e.g. HDRS -5.3 ± 4.0. I am able to calculate the outcome score (e.g. 11) but I need standard deviation data for the mean outcome scores, and am not clear if the data presented (e.g. ± 4.0) is the SD of the outcome scores, or in fact SD in the changes of between baseline and outcome scores. If it is the former it would be great if you could confirm this, and I can then use the data as is...If it is the latter I would need you to give me the SD data for the mean outcomes scores.

( I hope this is clear!)

Anyway, In summary I need:

Mean and standard deviation of baseline and outcome scores for both HDRS and BDI depression measures across all four conditions.

If you were able to clarify whether these are the data reported, or give me these data, it would be fantastic, and allow me to include your paper in my study.

Any help would be much appreciated.

Regards,

David Huxtable Specialist Psychological Practitioner Older Adult Psychology Dept Royal Cornhill Hospital,  
Aberdeen, AB25 2ZH  
(01224) 557 497

-No reply-

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8.

Huxtable David (NHS GRAMPIAN)

Sent: 01 May 2012 16:14

To: [stirling.moorey@slam.nhs.uk](mailto:stirling.moorey@slam.nhs.uk)

Subject: request for data: A cluster randomized controlled trial of cognitive behaviour therapy for common mental disorders in patients with advanced cancer

Hello,

I am undertaking a meta-analysis examining CBT for depression with older adults with co-morbid physical illness. This is in part-fulfillment of a Doctorate in Clinical Psychology at Edinburgh University.

Your paper: 'A cluster randomized controlled trial of cognitive behaviour therapy for common mental disorders in patients with advanced cancer' meets the inclusion criteria.

However, to include it in the meta-analysis, I would need intent-to treat mean outcome HADS depression scores with standard deviation for both CBT and Control conditions. If you were able to share this data it would be fantastic, as it would allow me to include your paper in my analysis.

Also, I was wondering if you were aware of any unpublished/current RCT trials examining CBT treatment for depression in older adults (>55yrs) with a physical illness, which I may not have been able to find via comprehensive literature searches of available databases.

Currently I have not found any other RCT trials looking at CBT for depression in patients with co-morbid cancer diagnoses with a mean age >55yrs. If you are aware of any it would be enormously helpful.

Many thanks,

David Huxtable Specialist Psychological Practitioner Older Adult Psychology Dept Royal Cornhill Hospital,  
Aberdeen, AB25 2ZH  
(01224) 557 497

-No reply-

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9.

Huxtable David (NHS GRAMPIAN)

Sent: 01 May 2012 15:34

To: [lustmanp@wustl.edu](mailto:lustmanp@wustl.edu)

Subject: FW: request for data re: Cognitive Behavior Therapy for Depression in Type 2 Diabetes Mellitus

Hello,

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I am undertaking a meta-analysis examining CBT for depression with older adults with co-morbid physical illness. This is in part-fulfillment of a Doctorate in Clinical Psychology at Edinburgh University.

Your paper: 'Cognitive Behavior Therapy for Depression in Type 2 Diabetes Mellitus' meets the inclusion criteria. However, I was wondering if you had mean BDI scores with standard deviation data at baseline, 10 weeks and 6 months, for both CBT and control groups. If you were able to give me this data, it would be great, and it would allow me to include the paper in my analysis.

Also, I was wondering if you were aware of any unpublished/current RCT trials examining CBT treatment for depression in older adults with a physical illness, which I would not have been able to find via comprehensive literature searches of available databases.

Any help would be much appreciated.

Regards,

David Huxtable Specialist Psychological Practitioner Older Adult Psychology Dept Royal Cornhill Hospital,  
Aberdeen, AB25 2ZH  
(01224) 557 497

-No reply-

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## Appendix 11: Full Search Strategy (Meta-Analysis).



1. EBSCO HOST: Last date searched: 8.4.2012

#	Query	Limiters/Expanders	Last Run Via	Results
S5	S1 and S2 and S4	Search modes - Boolean/Phrase	Interface - EBSCOhost Search Screen - Basic Search Database - CINAHL Plus with Full Text;MEDLINE with Full Text;PsycINFO;Psychology and Behavioral Sciences Collection;Biomedical Reference Collection: Comprehensive	2540
S4	Randomi?ed Controlled Trial OR RCT OR Controlled Trial	Search modes - Boolean/Phrase	Interface - EBSCOhost Search Screen - Basic Search Database - CINAHL Plus with Full Text;MEDLINE with Full Text;PsycINFO;Psychology and Behavioral Sciences Collection;Biomedical Reference Collection: Comprehensive	116885
S3	S1 and S2	Search modes - Boolean/Phrase	Interface - EBSCOhost Search Screen - Basic Search Database - CINAHL Plus with Full Text;MEDLINE with Full Text;PsycINFO;Psychology and Behavioral Sciences Collection;Biomedical Reference Collection: Comprehensive	39492
S2	psychotherap* OR cognitive therapy OR behavi* therapy OR CBT OR Problem Solving OR Stress Management	Limiters - Language: English; Age Groups: All Adult; Languages: English; Age Groups: Adulthood (18 yrs & older); Population Group: Human; Language: English Search modes - Boolean/Phrase	Interface - EBSCOhost Search Screen - Basic Search Database - CINAHL Plus with Full Text;MEDLINE with Full Text;PsycINFO;Psychology and Behavioral Sciences Collection;Biomedical Reference Collection: Comprehensive	245152
S1	depress* OR dysthymi* OR mood	Limiters - Language: English; Age Groups: All Adult; Languages: English; Age Groups: Adulthood (18 yrs & older); Population Group: Human; Language: English Search modes - Boolean/Phrase	Interface - EBSCOhost Search Screen - Basic Search Database - CINAHL Plus with Full Text;MEDLINE with Full Text;PsycINFO;Psychology and Behavioral Sciences Collection;Biomedical Reference Collection: Comprehensive	557622

2. COCHRANE LIBRARY: Last date searched: 16.5.2012



**THE COCHRANE LIBRARY**  
Independent high-quality evidence for health care decision making  
from The Cochrane Collaboration

SEARCH

Title, Abstract or Keywords

Advanced Search > MeSH Search > Search History > Saved Searches >

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**COCHRANE REVIEWS**

By Topic | New Reviews | Updated Reviews | A-Z | By Review Group

**OTHER RESOURCES**

Other Reviews | Trials | Methods Studies | Technology Assessments | Economic Evaluations

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**Search Results**

Show Results in:  
Trials [857]

There are 857 results out of 673964 records for: "(depress\* OR disthymi\* OR mood) in Title, Abstract or Keywords and randomi?ed controlled trial in Title, Abstract or Keywords and psychotherap\* OR psychosocial OR psychological OR cognitive OR behav\* OR stress in Title, Abstract or Keywords and cancer OR COPD OR diabetes OR heart OR dementia OR Alzheimer\* OR coronary OR Parkinson\* OR arthritis OR HIV OR chronic health OR physical \*morbidity OR multiple sclerosis OR irritable bowel OR physical illness OR epilepsy in Title, Abstract or Keywords in Cochrane Central Register of Controlled Trials"

View: 1-25 | 26-50 | 51-75 | 76-100 | 101-125 | Next >

[Export All Results](#)

3. OPEN GREY: Last date searched: 6.5.2012

Open Grey (<http://opensigle.inist.fr/> ) <http://www.opengrey.eu/>

Search terms:

(depress\* OR disthymi\* OR mood)

AND (psychotherap\* OR psychosocial OR psychological OR cognitive OR behav\* OR stress)

AND (cancer OR COPD OR diabetes OR heart OR dementia OR Alzheimer\* OR coronary OR Parkinson\* OR arthritis OR HIV OR chronic health OR physical \*morbidity OR multiple sclerosis OR irritable bowel OR physical illness)

AND (controlled trial)

Search results: 1

WORLD HEALTH ORGANISATION INTERNATIONAL CLINICAL TRIALS REGISTRY PLATFORM SEARCH PORTAL: Last date searched: 6.5.2012

<http://apps.who.int/trialsearch/default.aspx>

Search terms:

Title: depress\* OR disthymi\* OR mood

Condition: cancer OR COPD OR diabetes OR heart OR dementia OR Alzheimer\* OR coronary OR heart OR Parkinson\* OR arthritis OR HIV OR chronic health OR physical \*morbidity OR multiple sclerosis OR irritable bowel OR physical illness OR epilepsy

Intervention: psychotherap\* OR psychosocial OR psychological OR cognitive OR behav\* OR stress OR Problem Solving

Search results: 42

## Appendix 12: Excluded studies (Meta-Analysis).

Reference	Completed study	Mean age ≥55	RCT	Physical illness	Validated outcome measure	Mean base-line depression	Depression as focus of treatment and outcome	CBT?
Amsberg, S., Anderbro, T., Wredling, R., Lisspers, J., Lins, P.-E., Adamson, U., & Johansson, U.-B. (2009). A cognitive behavior therapy-based intervention among poorly controlled adult type 1 diabetes patients—a randomized controlled trial. <i>Patient education and counseling</i> , 77(1), 72-80. doi:10.1016/j.pec.2009.01.015	✓	✓ (41)	✓	✓	✓ HADS	HADS (D) 3.76	✓ (focus on self-care glycemic control)	✓
Andersson, G. (2002). Randomized Controlled Trial of Internet-Based Cognitive Behavior Therapy for Distress Associated With Tinnitus. <i>Psychosomatic Medicine</i> , 64(5), 810-816. doi:10.1097/01.PSY.0000031577.42041.F8	✓	✓ (48)	✓	✓	✓ HADS	HADS (D) 6.9	✓ (focus on tinnitus management)	✓
Areán, P. a. Raue, P. J., Macklin, R. S., Kanellopoulos, D., McCulloch, C., & Alexopoulos, G. S. (2010). Problem-Solving Therapy and Supportive Therapy in Older Adults With Major Depression and Executive Dysfunction. <i>American Journal of Psychiatry</i> , 167(11), 1391-1398. doi:10.1001/archgenpsychiatry.2010.177	✓	✓ (72)	✓	excludes co-morbidity dementia	✓ HDRS	HDRS 24.1	✓ (focus on stress)	✓ PST
Bastelaar, K. M. P. V., Pouwter, F., Cuijpers, P., Ripper, H., & Snoe, F. J. (2011). Web-Based Depression Treatment for Type 1 and Type 2 Diabetic Patients: A randomized controlled trial. <i>Diabetes Care</i> , 34, 320-325.	✓	✓ (50)	✓	✓	✓ CES-D	CES-D 28	✓ (focus on stress)	✓
Berger, S., Schadt, T., von Wyl, V., Ehler, U., Zellweger, C., Furrer, H., Regli, D., et al. (2008). Effects of cognitive behavioral stress management on HIV-1 RNA, CD4 cell counts and psychosocial parameters of HIV-infected persons. <i>AIDS (London, England)</i> , 22(6), 767-775. Department of Clinical Psychology and Psychotherapy, Institute of Psychology, University of Zurich, Switzerland.	✓	✓ (44)	✓	✓	✓ HADS	HADS 4.5 to 6.5	✓ (focus on stress)	✓
Berkman, L., Blumenthal, J., & Burg, M. (2003). Effects of Treating Depression and low perceived social support on clinical events after myocardial infarction: the Enhancing Recovery in Coronary Heart Disease Patients (ENRICHD) Randomized Trial. <i>JAMA: Journal of the American Medical Association</i> , 289(23), 3106-3116.	✓	✓ (81)	✓	✓	✓ BDI/HDRS	BDI/HDRS	✓ (focus on stress)	✓ CBT with AD
Blumenthal, J. A., Sherwood, A., Babyak, M. A., Watkins, L. L., Waugh, R., Georgiades, A., Bacon, S. L., et al. (2005). Effects of exercise and stress management training on markers of cardiovascular risk in patients with ischemic heart disease: a randomized controlled trial. <i>JAMA: Journal of the American Medical Association</i> , 293(13), 1626-1634. Department of Psychiatry and Behavioral Sciences, Duke University Medical Center, Box 3119, Durham, NC 27710.	✓	✓ (63)	✓	✓	✓ BDI	BDI 9.4	✓ (focus on stress)	✓
Boesen, E. H., ross, L., Frederiksen, K., Thomsen, B. L., Dahlstrøm, K., Schmidt, G., Næsted, J., et al. (2005). Psychoeducational intervention for patients with cutaneous malignant melanoma: a replication study. <i>Journal of Clinical Oncology</i> , 23(6), 1270-1277. Institute of Cancer Epidemiology, Danish Cancer Society, Strandboulevarden 49, DK-2100 Copenhagen, Denmark.	✓	✓ (est. 47)	✓	✓	✓ POMS	POMS-D 4.2	✓ (focus on coping and distress)	✓ (psycho-ed)
Bogner, H. R., Morales, K. H., Post, E. P., & Bruce, M. L. (2007). Diabetes, Depression, and Death: A randomized controlled trial of a depression treatment program for older adults in primary care (PROSPECT). <i>Diabetes Care</i> , 30(12), 3005.	✓	✓ (71)	✓	✓	✓ HDRS	HDRS (18)	✓ (diabetes management)	✓ (AD/IPT)
Bombardier, C. H., Bell, K. R., Temkin, N. R., Fann, J. R., Hoffman, J., & Dikmen, S. (2009). The efficacy of a scheduled telephone intervention for ameliorating depressive symptoms during the first year after traumatic brain injury. <i>Journal of Head Trauma Rehabilitation</i> , 24(4), 230-238. Departments of Rehabilitation Medicine, University of Washington School of Medicine, Seattle, USA. chb@u.washington.edu. doi:10.1097/HTR.0b013e3181ad65f0	✓	✓ (est. 36)	✓	✓	✓ BSI-D	BSI-D 0.58	✓ (main focus: diabetes management)	✓ (tel/contact)
Bond, G. E., Burr, R. L., Wolf, F. M., & Feldt, K. (2010). The effects of a web-based intervention on psychosocial well-being among adults aged 60 and older with diabetes: a randomized trial. <i>The Diabetes Educator</i> , 36(3), 446-56. doi:10.1177/0145721710366758	✓	✓ (68)	✓	✓	✓ CES-D	CES-D (12)	✓ (main focus: diabetes management)	✓
Brody, B. L., Roch-Leveccq, A. C., Kaplan, R. M., Moutier, C. Y., & Brown, S. I. (2006). Age-related macular degeneration: self-management and reduction of depressive symptoms in a randomized, controlled study. <i>Journal of the American Geriatrics Society</i> , 54(10), 1557-1562. Department of Ophthalmology, Shiley Eye Center, University of California at San Diego, La Jolla, CA 92093, USA. doi:10.1111/j.1532-5415.2006.00881.x	✓	✓ (81)	✓	✓	✓ GDS	GDS 7.5	✓ (main focus: diabetes management)	✓ (psycho-ed/problem solving)



Reference	Completed	Study	Mean age ≥55	RCT	Physical illness	Validated outcome measure	Mean base-line depression	depression as focus of treatment and outcome.	CBT?
Bruce, M., Have, T. T., & III, C. R. (2004). Reducing suicidal ideation and depressive symptoms in depressed older primary care patients. <i>JAMA: the Journal of</i> , 291(9), 1081-1091.	✓	✓	✓ (60-94)	✓	✗ (co-morb not detailed)	✓ HDRS	✓ HDRS 18.6	✓	✗ (Care management)
Ciechanowski, P., Chaytor, N., Miller, J., Frasier, R., Russo, J., Unutzer, J., & Gilliam, F. (2010). PEARLS depression treatment for individuals with epilepsy: a randomized controlled trial. <i>Epilepsy &amp; Behavior: E&amp;B</i> , 29(3), 225-231. Department of Psychiatry and Behavioral Sciences, University of Washington, Seattle, WA 98195-6560. USA. pavelic@u.washington.edu.	✓	✓	✗ (31)	✓	✓	✓ HSCL-20	✓ HSCL-20 2	✓	✗ (PST with Care management)
Ciechanowski, P., Wagner, E., Schmalzer, K., Schwartz, S., Diehr, P., Kulzer, J., Gray, S. (2004). Community-Integrated Home-Based Depression Treatment in Older Adults. <i>291(13)</i> , 1569-1577.	✓	✓	✓ (74)	✓	✓ (4.6 (SD, 2.1) chr cond.)	✓ HSCL-20	✓ HSCL-20 1.2	✓	✗ (PST with Care management/AD meds)
Claesson, M., Birgander, L. S., Lindahl, B., Nasic, S., Aström, M., Asplund, K., & Burell, G. (2005). Women's hearts--stress management for women with ischemic heart disease: explanatory analyses of a randomized controlled trial. <i>Journal of cardiopulmonary rehabilitation</i> , 25(2), 93-102.	✓	✓	✓ (59)	✓	✓	✓ CPRSA 5.5	✓ CPRSA 5.5	✓	✗ (focus on stress)
Cosio, D., Jin, L., Siddique, J., & Mohr, D. C. (2011). The effect of telephone-administered cognitive-behavioral therapy on quality of life among patients with multiple sclerosis. (2012) <i>Annals of Behavioral Medicine: A Publication Of The Society Of Behavioral Medicine</i> , 41(2), 227-234.	✓	✓	NR	✓	✓	✓ BDI	✓ BDI >18	✓	✓ (depressive symptoms)
Craig, A. R., Hancock, K., Dickson, H., & Chang, E. (2007). Long-Term Psychological Outcomes in Spinal Cord Injured Persons: Results of a Controlled Trial Using Cognitive Behavior Therapy. <i>78(January 1997)</i> , 33-38.	✓	✓	✗ (44)	✗	✓	✓ BDI	✓ BDI 11.6	✓	✓
Craigie, M. A., & Nathan, P. (2009). A nonrandomized effectiveness comparison of broad-spectrum group CBT to individual cbt for depressed outpatients in a community mental health setting. <i>Behavior Therapy</i> , 40(3), 302-314. Craigie, Mark A., Centre for Clinical Interventions, Perth, Australia. 6003. markcraigie@bigpond.com.au: Elsevier Science.	✓	✓	✗ (36)	✗	✗	-	-	✓	-
Davidson, K. W., Rieckmann, N., Clemow, L., Schwartz, J. E., & Shimo, D. (2010). Enhanced Depression Care for Patients With Acute Coronary Syndrome and Persistent Depressive Symptoms. <i>Archives of Internal Medicine</i> , 170(7), 600-608.	✓	✓	✓ (81)	✓	✓	✓ BDI	✓ BDI ≥10	✓	✗ stepped care inc mix of PST and AD.
de Godoy, D. V., & de Godoy, R. F. (2003). A randomized controlled trial of the effect of psychotherapy on anxiety and depression in chronic obstructive pulmonary disease. <i>Archives of Physical Medicine and Rehabilitation</i> , 84(8), 1154-1157. doi:10.1016/S0003-9993(03)00239-9	✓	✓	✓ (60)	✓	✓	✓ BDI	✓ BDI 13.71	✓	✗ (elements)
Doering, L. V., Cross, R., Vredevoe, D., Martinez-maza, O., & Cowan, M. J. (2007). Infection, depression, and immunity in women after coronary artery bypass: a pilot study of cognitive behavioral therapy. <i>Alternative Therapies in Health And Medicine</i> , 13(3), 18-20.	✓	✓	✓ (63.6)	✗	✓	✓ DISH	-	✓	✓
Drossman, D. a, Toner, B. B., Whitehead, W. E., Diamant, N. E., Dalton, C. B., Duncan, S., et al. (2003). Cognitive-behavioral therapy versus education and desipramine versus placebo for moderate to severe functional bowel disorders. This study was registered with ClinicalTrials.gov (trial registry no. NCT00006157). <i>Gastroenterology</i> , 125(1), 19-31. doi:10.1016/S0016-5085(03)00669-3	✓	✓	✗ (38)	✓	✓	✗	-	✓	✗ (main focus: illness management)
Duarte, P. S., Miyazaki, M. C., Blay, S. L., & Sessa, R. (2009). Cognitive-behavioral group therapy is an effective treatment for major depression in hemodialysis patients. <i>Kidney International</i> , 76(4), 414-21. doi:10.1038/ki.2009.156	✓	✓	✗ (52)	✓	✓	✓ BDI	✓ BDI 24.2	✓	✓

Reference	Completed study	Mean age ± SD	RCT	Physical illness	Validated outcome measure	Mean base-line depression	Depression as focus of treatment and outcome	CBT?
Ell, K., Aranda, M. P., Xie, B., Lee, P.-J., & Chou, C.-P. (2010). Collaborative depression treatment in older and younger adults with physical illness: pooled comparative analysis of three randomized clinical trials. <i>The American Journal of Geriatric Psychiatry</i> . <i>official Journal of the American Association for Geriatric Psychiatry</i> , 18(6), 520-30. doi:10.1097/JGP.0b013e3181cc0350	✓	✓ (71.6)	✓	✓	✓ PHQ-9	PHQ-9 ≥10	✓	✓ (PST/coll care)
Evers, A. W. M., Kraaijmaat, F. W., van Riel, P. L. C. M., & de Jong, A. J. L. (2002). Tailored cognitive-behavioral therapy in early rheumatoid arthritis for patients at risk: a randomized controlled trial. <i>Pain</i> , 100(1-2), 141-153. Department of Medical Psychology, University Medical Center St Radboud, P. O. Box 9101, 6500 HB Nijmegen, The Netherlands. a.evers@cuk.zumcn.nl.	✓	✓ (54)	✓	✓	✓ BDI	-	✓ 1 module of 4: mood	✓
Forman, A. C., & Lincoln, N. B. (2010). Evaluation of an adjustment group for people with multiple sclerosis: a pilot randomized controlled trial. <i>Clinical rehabilitation</i> , 24(3), 211-21. doi:10.1177/0269215509343492	✓	✓ (47.7)	✗	✓	✓ HAD S	HADS ≥7	✗	✗
Fukui, S., Kugaya, A., Okamura, H., & Kamiya, M. (2000). A psychosocial group intervention for Japanese women with primary breast carcinoma. <i>Cancer</i> , 1026-1036.	✓	✓ (52)	✓	✓	✓ HADS D	HADS D 4.5 illness management)	✗ (psychosocial management)	✗
Garnefski, N., & Kraaij, V. (2012). Effects of a Cognitive Behavioral Self-help program on emotional problems for people with acquired hearing loss: a randomized controlled trial. <i>Journal of deaf studies and deaf education</i> , 17(1), 75-84. doi:10.1093/deaf/edm020	✓	✓ (57)	✓	✓	✓ HADS 7.07	HADS_D	✓ focused on anxiety and depression	✗
Gellis, Z. (2010). Problem solving therapy for subthreshold depression in home healthcare patients with cardiovascular disease. <i>The American Journal of geriatric psychiatry</i> ; (June), 464-474.	✗ (pill not)	✓ (76)	✓	✓	✓ BDI/H AM-D	BDI 27 / HAM_D 18	✓	✓ PST
Gensichen, J. (2006). IMPACT collaborative care improves depression in elderly patients in primary care in the longer term. <i>Evidence Based Mental Health</i> , 9(3), 76. Institute for General Practice, Chronic Care and Health Services Research Center, Johann Wolfgang Goethe University Hospital Frankfurt, Germany.	✗	-	-	-	-	-	-	-
Giesler, R. B., Given, C. W., Rawl, S., Monahan, P., Burns, D., Azzouz, F., et al. (2005). Improving the quality of life of patients with prostate carcinoma: a randomized trial testing the efficacy of a nurse-driven intervention. <i>Cancer</i> , 104(4), 752-762. School of Nursing, Indiana University, Indianapolis, Indiana, USA. bgiesler@iupui.edu.	✓	✓ (67)	✓	✓	✓ CES-D	CES-D 6.9	✗ (general focus on QOL/adjustment)	✓
Greer, S., Moorey, S., Baruch, J., & Watson, M. (1992). Adjuvant psychological therapy for patients with cancer: a prospective randomised trial. <i>British Medical</i> .	✓	✗ (51)	✓	✓	✓ HAD S	HADS 6.2	✗	✓
Harpole, L. H., Williams Jr, J. W., Olsen, M. K., Stechuchak, K. M., Oddone, E., Callahan, C. M., Katon, W. J., et al. (2005). Improving depression outcomes in older adults with comorbid medical illness. <i>General Hospital Psychiatry</i> , 27(1), 4-12. Department of Medicine, Duke University Medical Center, Durham, NC 27709, USA. Linda.harpole@gsk.com.	✓	✓	✓	✓ (mean 3.8 Chr HC)	✓ SCL-20	-	✓	✗ (PST/coll care)
Heckman, T. G., Sikkema, K. J., Hansen, N., Kochman, A., Heh, V., & Neufeld, S. (2011). A randomized clinical trial of a coping improvement group intervention for HIV-infected older adults. <i>Journal of behavioral medicine</i> , 34(2), 102-11. doi:10.1007/s10865-010-9292-6	✓	✓ (55.3)	✓	✓	✓ GDS	(0-9 = 42%; 10-19 = 38%; 20-30 = 22%)	✗	✗
Hedayati, S. S., Yalamanchili, V., & Finkelstein, F. O. (2012). A practical approach to the treatment of depression in patients with chronic kidney disease and end-stage renal disease. <i>Kidney International</i> , 81(3), 247-255. doi:10.1038/ki.2011.358.A	✓	✗	✗	-	(summary paper)	-	-	-



Reference	Completed study	Mean age ≥55	RCT	Physical illness	Validated outcome measure	Mean base-line depression	Depression as focus of treatment and outcome	CBT?
Hewlett, S., Ambler, N., Almeida, C., Cliss, A., Hammond, A., Kitchen, K., Knops, B., et al. (2011). Self-management of fatigue in rheumatoid arthritis: a randomised controlled trial of group cognitive-behavioural therapy. <i>Annals Of The Rheumatic Diseases</i> , 70(6), 1060-1067. Department of Nursing and Midwifery, University of the West of England, Bristol, UK. Sarah.Hewlett@uwe.ac.uk	✓	✓ (59.2)	✓	✓	✓	-	✓	✓
Irvine, J., Firestone, J., Ong, L., Cribbie, R., Dorian, P., Harris, L., Ritvo, P., et al. (2011). A randomized controlled trial of cognitive behavior therapy tailored to psychological adaptation to an implantable cardioverter defibrillator. <i>Psychosomatic medicine</i> , 73(3), 226-33. doi:10.1097/PSY.0b013e31820af6c3	✓	✓ (64.4)	✓	✓	✓ HAD S-D	-	✓ (psychological adaptation)	✓
Irvine, J., Firestone, J., Ong, L., Cribbie, R., Dorian, P., Harris, L., Ritvo, P., et al. (2011). A randomized controlled trial of cognitive behavior therapy tailored to psychological adaptation to an implantable cardioverter defibrillator. <i>Psychosomatic Medicine</i> , 73(3), 226-233. DPhil, Department of Psychology, York University, Behavioural Sciences Building, 4700 Keele Street, Toronto, Ontario, Canada, M3J 1P3. jirvine@yorku.ca. doi:10.1097/PSY.0b013e31820af6c3	✓	✓ (66)	✓	✓	✓ HADS 4.12	HADS-D	✓ (based on cognitive theory of anxiety: focus on coping/adjustment)	✓
Ismail, K., Maissi, E., Thomas, S., Chalder, T., Schmidt, U., Bartlett, J., Patel, a., et al. (2010). A randomised controlled trial of cognitive behaviour therapy and motivational interviewing for people with Type 1 diabetes mellitus with persistent sub-optimal glycaemic control: a Diabetes and Psychological Therapies (ADAPT) study. <i>Health technology assessment (Winchester, England)</i> , 14(22), 1-101, iii-iv. doi:10.3310/ht14220	✓	✓ (36)	✓	✓	✓	-	✓	✓
Jonkers, Catharina CM, Lamers, F., Bosma, H., Metsemakers, J. F. M., & van Eijk, J. T. M. (2012). The effectiveness of a minimal psychological intervention on self-management beliefs and behaviors in depressed chronically ill elderly persons: a randomized trial. <i>International Psychogeriatrics // PA</i> , 24(2), 288-297. Department of Social Medicine, School for Public Health and Primary Care (Caphri), Maastricht University, Maastricht, The Netherlands. jonkers@zonmw.nl	✓	✓ (60+)	✓	✓	✓	-	✓	✓
Katon, W. J., Korff, M. V., Lin, E. H. B., Simon, G., Ludman, E., Russo, J., Ciechanowski, P., et al. (2004). A Randomized Trial of Collaborative Care in Patients With Diabetes and Depression. <i>Archives of General Psychiatry</i> , 61, 1042-1049.	✓	✓ (58.1)	✓	✓	✓ PHQ -9/SCC-PHQ-9 ≥10	-	✓	✓ Coll Care (PST alone = 7.9%)
Kennedy, P., Duff, J., Evans, M., & Beedie, A. (2003). Coping effectiveness training reduces depression and anxiety following traumatic spinal cord injuries. <i>British Journal of Clinical Psychology</i> , 42(1), 41-52. Kennedy, P., Dept of Clinical Psychology, National Spinal Injuries Ctr, Stoke Mandeville Hosp NHS Trust, Aylesbury, BKM, United Kingdom, HP21 8AL, paul.kennedy@smh.nhs.uk: British Psychological Society.	✓	✓ (34.7)	✓	✓	✓ BDI	-	✓	✓ (elements)
Kerse, N., Hayman, K., & Moyes, S. (2010). Home-Based Activity Program for Older People With Depressive Symptoms: DeLUTE—A Randomized Controlled Trial. <i>The Annals of Family</i> , 214-224. doi:10.1370/afm.1093.INTRODUCTION	✓	✓ (81)	✓	✓	✓ GDS	GDS 7.4	✓ (main focus: activity/functioning)	✓
King, C., & Kennedy, P. (1999). Coping effectiveness training for people with spinal cord injury: preliminary results of a controlled trial. <i>The British Journal of clinical psychology / The British Psychological Society</i> , 38 ( Pt 1), 5-14.	✓	✓ (35.8)	✓ (CT)	✓	✓ BDI	BDI 15.5	✓	✓ (elements)
Kinsinger, S. W., Lattie, E., & Mohr, D. C. (2011). Relationship between depression, fatigue, subjective cognitive impairment, and objective neuropsychological functioning in patients with multiple sclerosis. <i>Neuropsychology</i> , 24(5), 573-580. doi:10.1037/a0019222.Relationship	✓	✓ (47)	✓	✓	✓ BDI	BDI ≥16	✓	✓ (tel)
Kiosses, D. N., Areal, P. a, Teri, L., & Alexopoulos, G. S. (2010). Home-delivered problem adaptation therapy (PATH) for depressed, cognitively impaired, disabled elders: A preliminary study. <i>The American journal of geriatric psychiatry : official journal of the American Association for Geriatric Psychiatry</i> , 18(11), 988-99. doi:10.1097/JGP.0b013e3181d6947d	✓ (pill not)	✓ (80)	✓	✓	✓ HAM-D24	HAM-D24 22.4	✓	✓ PST
Kissane, D. W., Bloch, S., Smith, G. C., Mlach, P., Clarke, D. M., Ikin, J., Love, A., et al. (2003). Cognitive-existent group psychotherapy for women with primary breast cancer: a randomised controlled trial. <i>Psycho-oncology</i> , 12(6), 532-46. doi:10.1002/pon.683	✓	✓ (44)	✓	✓	✓ HADS	HADS-D 3.7	✓ adjustment and general psychosocial outcomes	✓ some key cognitive techniques

Reference	Completed study	Mean age ≥55	RCT	Physical illness	Validated measure	Mean base-line depression	depression as focus of treatment and outcome	CBT?
Korstjens, I., Mesters, I., May, A. M., van Weert, E., van den Hout, J. H. C., Ros, W., Hoekstra-Weebers, J. E. H. M., et al. (2010). Effects of cancer rehabilitation on problem-solving, anxiety and depression: A RCT comparing physical and cognitive-behavioural training versus physical training. <i>Psychology &amp; health</i> , (April 2012), 1-20. doi:10.1080/08870441.003611569	✓	✓ (49)	✓	✓	✓ HAD S-D	-	✓	✓
Kraaij, V., van Emmerik, A., Garnefski, N., Schroevers, M. J., Lo-Fo-Wong, D., van Empelen, P., Dusseldorp, E., et al. (2010). Effects of a cognitive behavioral self-help program and a computerized structured writing intervention on depressed mood for HIV-infected people: a pilot randomized controlled trial. <i>Patient Education &amp; Counseling</i> , 80(2), 200-204. Department of Medical Psychology, Leiden University Medical Center, Leiden, The Netherlands. V.Kraaij@lumc.nl. doi:10.1016/j.pec.2009.08.014	✓	✓ (49)	✗	✓	✓ HAD S-D	HADS-D 7.31-8.13	✓	✗ (elements)
Kraaijmaat, F., Brons, M., & Geenen, R. (1995). The effect of cognitive behavior therapy in patients with rheumatoid arthritis. <i>Behaviour research and</i> , 33(5), 487-495.	✓	✓ (57)	✓	✓	✗ IRGL	-	✗ (treatment focus on pain management)	✓
Kunik, M. E., Braun, U., Stanley, M. a, Wristers, K., Molinari, V., Stoeberl, D., & Orengo, C. a. (2001). One session cognitive behavioural therapy for elderly patients with chronic obstructive pulmonary disease. <i>Psychological medicine</i> , 31(4), 717-23.	✓	✓ (73)	✓	✓	✓ GDS	GDS 7.7-11.5	✗ (treatment focus on anxiety)	✓
Lamers, Femke, Jonkers C.M., C., Bosma, H., Knottnerus André, J., & Th. M. (2011). Treating depression in diabetes patients: does a nurse-administered minimal psychological intervention affect diabetes-specific quality of life and glycaemic control? A randomized controlled trial. <i>Journal of Advanced Nursing</i> , 67(4), 788-799. Researcher Department of Social Medicine, School for Public Health and Primary Care (CAPRI), Maastricht University, The Netherlands. doi:10.1111/j.1365-2648.2010.05540.x	✓	✓ (70)	✓	✓	✗	-	✓	✗
Lamers, Femke, Jonkers, C. M., Bosma, H., Kempen, G. I. J. M., Meijer, J. a M. J., Penninx, B. W. J. H., Knottnerus, J. A., et al. (2010). A minimal psychological intervention in chronically ill elderly patients with depression: a randomized trial. <i>Psychotherapy and psychosomatics</i> , 79(4), 217-26. doi:10.1159/000313690	✓	✓ (71)	✓	✓	✓ BDI/HS DRS	BDI 17, HDRS 13-14	✓	✗ (minimal intervention)
Lefort, S. (2001). Cognitive behavioural therapy plus medical management reduced depression and joint inflammation in rheumatoid arthritis. <i>Evidence Based Nursing</i> , 4(4), 120. Associate Professor, School of Nursing, Memorial University of Newfoundland, St John's, Newfoundland, Canada.	✗ review							
Lin, Elizabeth H B, Katon, W., Williams, J. W., Kroenke, K., Hunkeler, E., Harpole, L., Hoffing, M., et al. (2003). Effect of Improving Depression Care on Pain and Functional Outcomes. <i>JAMA : the journal of the American Medical Association</i> , 290(18).	✓	✓ (72)	✓	✓	✓ HSC	16% MajD, 29% Dysth	✗	✗ (coll care)
Lincoln, N B, Dent, A., Harding, J., Weyman, N., Nicholl, C., Blumhardt, L. D., & Playford, E. D. (2002). Evaluation of cognitive assessment and cognitive intervention for people with multiple sclerosis. <i>Journal of Neurology, Neurosurgery &amp; Psychiatry</i> , 72(1), 93-98. Lincoln, N. B., School of Psychology, University of Nottingham, University Park, Nottingham, United Kingdom, NG7 2RD, nbl@psychology.nottingham.ac.uk; BMJ Publishing Group.	✓	✗ (40)	✓	✓	✗	-	✗	✗
Lincoln, Nadina B, Yull, F., Holmes, J., Drummond, A. E. R., Constantinescu, C. S., Armstrong, S., & Phillips, C. (2011). Evaluation of an adjustment group for people with multiple sclerosis and low mood: a randomized controlled trial. <i>Multiple Sclerosis (Houndmills, Basingstoke, England)</i> , 17(10), 1250-1257. Institute of Work, Health and Organisations, University of Nottingham, UK. nadina.lincoln@nottingham.ac.uk	✓	✗ (44)	✓	✓	✓ BDI	BDI 21-23	✗	✓
Ljótsson, B., Falk, L., Vesterlund, A. W., Hedman, E., Lindfors, P., Ruck, C., Hursti, T., et al. (2010). Internet-delivered exposure and mindfulness based therapy for irritable bowel syndrome--a randomized controlled trial. <i>Behaviour research and therapy</i> , 48(6), 531-9. Elsevier Ltd. doi:10.1016/j.brat.2010.03.003	✓	✗ (35)	✓	✓	✓ MADRS-S	MADRS-S 12.5	✗ (main focus: illness management)	✓
Lorig, K. R., Sobel, D. S., Ritter, P. L., Laurent, D., & Hobbs, M. (2001). Effect of a self-management program on patients with chronic disease. <i>Effective Clinical Practice</i> , 4, 256-262.	✓	✓ (62)	✓	✓	✗		✗	✗



Reference	Completed study	Mean age ± SD	RCT	Physical illness	Validated outcome measure	Mean base-line depression	Depression as focus of treatment and outcome	CBT?
Mohr, D. C., Hart, S., & Vella, L. (2007). Reduction in disability in a randomized controlled trial of telephone-administered cognitive-behavioral therapy. <i>Health Psychology, 26</i> (5), 554-563. Department of Psychiatry, University of California, USA. dmohr@northwestern.edu.	✓	✓ (47-49)	✓	✓	✓ HDRS/ BDI	✓	✓	✓
Mohr, D. C., Hart, S. L., Julian, L., Catledge, C., Honos-Webb, L., Vella, L., & Tasch, E. T. (2005). Telephone-administered psychotherapy for depression. <i>Archives Of General Psychiatry, 62</i> (9), 1007-1014.	✓	✓ (48.6)	✓	✓	✓ BDI-II	✓ BDI-II (27)	✓	✓
Navarrete-Navarrete, N., Peralta-Ramirez, M. I., Sabio-Sánchez, J. M., Coin, M. a. Robles-Ortega, H., Hidalgo-Tenorio, C., Ortega-Centeno, N., et al. (2010). Efficacy of cognitive behavioural therapy for the treatment of chronic stress in patients with lupus erythematosus: a randomized controlled trial. <i>Psychotherapy and psychosomatics, 79</i> (2), 107-115. doi:10.1159/000276370	✓	✓ (40-43)	✓	✓	✓ BDI	✓ BDI 14-16	✓	✓
Nickel, M. K., Lahmann, C., Muehlbacher, M., Nickel, C., Pedrosa Gil, F., Buschmann, W., Rother, N., et al. (2006). Change in instrumental activities of daily living disability in female senior patients with musculoskeletal pain: a prospective, randomized, controlled trial. <i>Archives Of Gerontology And Geriatrics, 42</i> (3), 247-255. Clinic for Psychiatry and Psychosomatic Medicine, Imntalklinik, D-84359 Simbach/Inn, Germany. m.nickel@imntalklinik.de.	✓	✓ (75)	✓	✓ (pain)	✓ CES-D	✓ CES-D 32-33	✓	✓
Nordin, L., & Rorsman, I. (2012). Cognitive behavioural therapy in multiple sclerosis: a randomized controlled pilot study of acceptance and commitment therapy. <i>Journal Of Rehabilitation Medicine: Official Journal Of The UEMS European Board Of Physical And Rehabilitation Medicine, 44</i> (1), 87-90. Department of Neurology, Skane University Hospital-Lund, Lund, Sweden.	✓	✓ (43-48)	✓ (Pit)	✓	✓ HADS- D/BDI	✓ HADS-D 5-7/BDI 13-15	✓	✓
Payne, a., & Blanchard, E. B. (1995). A controlled comparison of cognitive therapy and self-help support groups in the treatment of irritable bowel syndrome. <i>Journal of consulting and clinical psychology, 63</i> (5), 779-86.	✓	✓ (40)	✓	✓	✓ BDI	✓ BDI 14.6	✓	✓ (main focus: illness management)
Pitceathly, C., Maguire, P., Fletcher, J., Parle, M., Tomenson, B., & Creed, F. (2009). Can a brief psychological intervention prevent anxiety or depressive disorders in cancer patients? A randomised controlled trial. <i>Annals Of Oncology: Official Journal Of The European Society For Medical Oncology / ESMO, 20</i> (5), 928-934.	✓	✓ (50)	✓	✓	✓ HADS	✓	✓	✓ prevention: coping skills general coping emphasis
Raj, a. (2004). PEARLS home based treatment significantly improves depression, dysthymia, and health related quality of life in older people. <i>Evidence-Based Mental Health, 7</i> (4), 110-110. doi:10.1136/ebmh.7.4.110	✓	✓	✓	Summary of Ciecchanowski et al (2004)	✓	✓	✓	✓
Reme, S. E., Kennedy, T., Jones, R., Darnley, S., & Chalder, T. (2010). Predictors of treatment outcome after cognitive behavior therapy and antidepressant treatment for patients with irritable bowel syndrome in primary care. <i>Journal Of Psychosomatic Research, 68</i> (4), 385-388. Department of Psychological Medicine, King's College London, UK. silje.reme@uib.no.	✓	✓ (33-8)	✓	✓	✓	✓	✓	✓
Rigby, S. a. Thornton, E. W., & Young, C. a. (2008). A randomized group intervention trial to enhance mood and self-efficacy in people with multiple sclerosis. <i>British journal of health psychology, 13</i> (Pt 4), 619-31. doi:10.1348/135910707X241505	✓	✓ (44)	✓	✓	✓ HADS	✓ HADS 5	✓	✓ (coping)
Robinson, R., Jorge, R., & Moser, D. (2008). Escitalopram and problem-solving therapy for prevention of poststroke depression. <i>JAMA: the journal of, 299</i> (20).	✓	✓ (61-67)	✓	✓	✓ HDRS	✓ (prevention)	✓	✓ (prevention)
Robinson, S. K., Vliere, E. S., Bailey, K. A., Kindermann, S., Minassian, A. L., Goldin, P. R., Pedrelli, P., et al. (2008). A randomized controlled trial of cognitive-behavior therapy for tinnitus. <i>The International Tinnitus Journal, 14</i> (2), 119-126. University of California-San Diego Department of Psychiatry, San Diego, California 92161, USA. skrobinson@ucsd.edu.	✓	✓ (55)	✓	✓ (tinnitus)	✓ HDRS/ BDI	✓ HDRS 11-13/BDI 13-16	✓	✓ (focus on tinnitus management)

Reference	Completed study	Mean age ≥55	RCT	Physical illness	Validated outcome measure	Mean base-line depression	Depression as focus of treatment and psychosocial factors	CBT?
Ross, L., Thomsen, B. L., Karlsen, R. V., Boesen, E. H., & Johansen, C. (2005). A randomized psychosocial intervention study on the effect of home visits on the well-being of Danish colorectal cancer patients—the INCA project. <i>Psycho-Oncology</i> , 14(11), 949–961. doi:10.1002/pon.899	✓	✓ (69)	✓	✓	✓ HADS -	BDI 17.8	✓ (general focus on psychosocial factors)	✓ home visits
Saab, P. G., Bang, H., Williams, R. B., Powell, L. H., Schneiderman, N., Thoresen, C., Burg, M., et al. (2009). The impact of cognitive behavioral group training on event-free survival in patients with myocardial infarction: the ENRICHD experience. <i>Journal Of Psychosomatic Research</i> , 67(1), 45–56.	✓	✓ (61)	✓	✓	✓ BDI	BDI 17.8	✓	✓
Saffren, S. a, O' Cleirigh, C., Tan, J. Y., Raminani, S. R., Reilly, L. C., Otto, M. W., & Mayer, K. H. (2009). A randomized controlled trial of cognitive behavioral therapy for adherence and depression (CBT-AD) in HIV-infected individuals. <i>Health psychology: official journal of the Division of Health Psychology, American Psychological Association</i> , 28(1), 1–10. doi:10.1037/a0012715	✓	✗ (not explic it)	✓	✓	✓ HAIM- D, BDI	✓ HAIM- HAM-D18, BDI 24	✓	✓
SAVARD, J., SIMARD, S., GIGUERE, I., IVERS, H., MORIN, C. M., MAUNSELL, E., GAGNON, P., et al. (2006). Randomized clinical trial on cognitive therapy for depression in women with metastatic breast cancer: psychological and immunological effects. <i>and Supportive Care</i> , 4(219–237).	✓	✓ (51/ 52)	✓	✓	✓ BDI/H BDI ADS	21/HADS 9	✓	✓
Schmaling, K. B., Williams, B., Schwartz, S., Ciechanowski, P., & LoGerfo, J. (2008). The content of behavior therapy for depression demonstrates few associations with treatment outcome among low-income, medically ill older adults. <i>Behavior Therapy</i> , 39(4), 360–365. University of North Carolina at Charlotte, College of Health and Human Services, 9201 University City Blvd., Charlotte, NC 28223, USA. kbschmal@uncc.edu.	✓	✓ (72 6)	✗	✓ (4.7 chrillm)	✓ HSCE- 20	✓ HSCE- HSCE-20 1.31	✓	✗
Schwarz, P., & Blanchard, B. (1991). Evaluation of a psychological treatment for inflammatory bowel disease. <i>Behaviour Research and Therapy</i> , 29(2), 167–177.	✓	✗ (24- 38)	✗ (pit)	✓ (IBS)	✓ BDI	-	✗	✗
Sharpe, L., Sensky, T., Timberlake, N., Ryan, B., & Allard, S. (2003). Long-term efficacy of a cognitive behavioural treatment from a randomized controlled trial for patients recently diagnosed with rheumatoid arthritis. <i>Rheumatology</i> , 42(3), 435–441. doi:10.1093/rheumatology/keg144	✓	✓ (55- 1)	✓	✓	✓ HAD- D	✓ HAD- HAD-D 5.1- 5.3	✗ (focus on anxiety)	✓
Sharpe, L., Sensky, T., Timberlake, N., Ryan, B., Brewin, C. R., & Allard, S. (2001). A blind, randomized, controlled trial of cognitive-behavioural intervention for patients with recent onset rheumatoid arthritis: preventing psychological and physical morbidity. <i>Pain</i> , 89(2–3), 275–83.	✓	✓ (54 -57)	✓	✓	✓ HAD- D	✓ HAD- HAD-D 4.7- 4.9	✗ (focus on anxiety)	✓
Snoek, F. J., van der Ven, N. C. W., Twisk, J. W. R., Hogenelst, M. H. E., Tromp-Wever, a M. E., van der Ploeg, H. M., & Heine, R. J. (2008). Cognitive behavioural therapy (CBT) compared with blood glucose awareness training (BGAT) in poorly controlled Type 1 diabetic patients: long-term effects on HbA1c moderated by depression. A randomized controlled trial. <i>Diabetic medicine: a Journal of the British Diabetic Association</i> , 25(11), 1337–42. doi:10.1111/j.1464-5491.2008.02595.x	✓	✗ (38.1)	✓	✓	✓ CES-D	✓ CES-D CES-D, 16	✗ (main focus: illness management)	✓
Stiefel, F., Zdrojewski, C., & Hadj, F. B. (2008). Effects of a multifaceted psychiatric intervention targeted for the complex medically ill: a randomized controlled trial. <i>Psychotherapy and Psychosomatics</i> , 247–256. doi:10.1159/00012	✓	NR	✓	✓	✓ CES-D	✓ CES-D 26- 27	✗	✗
Tesar, N., Bandion, K., & Baumhackl, U. (2005). Efficacy of a neuropsychological training programme for patients with multiple sclerosis -- a randomised controlled trial. <i>Wiener Klinische Wochenschrift</i> , 117(21–22), 747–754. Department of Neurology, St. Pölten Regional Hospital, St. Pölten, Austria. mnt@gmx.at.	✓	✗ (45- 3)	✓	✓	✓ BDI	BDI 10–11	✗	✗
Thornton, L. M., Andersen, B. L., Schuler, T. a, & Carson, W. E. (2009). A psychological intervention reduces inflammatory markers by alleviating depressive symptoms: secondary analysis of a randomized controlled trial. <i>Psychosomatic medicine</i> , 71(7), 715–24. doi:10.1097/PSY.0b013e3181b0545c	✓	✗ (50)	✓	✓	✓ CES-D	✓ CES-D 12	✗	✗ (elements)



Reference	Completed study	Mean age ≥55	RCT	Physical illness	Validated outcome measure	Mean base-line depression measure	depression as focus of treatment and outcome.	CBT?
Tsay, S.-L., & Hung, L.-O. (2004). Empowerment of patients with end-stage renal disease—A randomized controlled trial. <i>International Journal of Nursing Studies</i> , 41(1), 59-65. Tsay, Shioh-Luan, Graduate Institute of Nursing, National Taipei College of Nursing 365 Ming Te Road, Pei-Tou, Taipei, Taiwan, sltsay@ntcn.edu.tw; Elsevier Science.	✓	NR	✓	✓	✓BDI	BDI 12.2	✗	✗
Turner, J. a, Mand, L., & Aaron, L. a. (2006). Short- and long-term efficacy of brief cognitive-behavioral therapy for patients with chronic temporomandibular disorder pain: a randomized, controlled trial. <i>Pain</i> , 122(3), 181-94. doi:10.1016/j.pain.2005.11.017	✓	✗ (35-38)	✓	✓	✓BDI	BDI 13.4	✗	✓
van Bastelaar KMP, Pouwer F, Cuijpers P, Riper H, Twisk JWR, S. F. (2012). Effect Modifier in a Web-Based Depression Treatment for Adults With Type 1 or Type 2 Diabetes? Secondary Analyses From a Randomized Controlled Trial. <i>J Med Internet Res</i> , 14(1:e2), doi: 10.2196/jmir.1657.	✗ Refer to van Bastelaar et al., (2011)							
Wetherell, J. L., Afari, N., Rutledge, T., Sorrell, J. T., Stoddard, J. a, Petkus, A. J., Solomon, B. C., et al. (2011). A randomized, controlled trial of acceptance and commitment therapy and cognitive-behavioral therapy for chronic pain. <i>Pain</i> , 152(9), 2098-107. International Association for the Study of Pain. doi:10.1016/j.pain.2011.05.016	✓	✓(55)	✗	✗ (pain)	✓BDI	BDI 15-19	✗ (focus on pain management)	✓
Zedlitz, A. M. E., Rietveld, T. C. M., Geurts, A. C., & Fasotti, L. (2012). Cognitive and graded activity training can alleviate persistent fatigue after stroke: a randomized, controlled trial. <i>Stroke: a journal of cerebral circulation</i> , 43(4), 1046-51. doi:10.1161/STROKEAHA.111.632117	✓	✓(55)	✓	✓	✓HAD-D	HAD-D 6.6-7.7	✗ (fatigue)	✓

Papers meeting inclusion criteria but excluded due to insufficient data

Reference	Completed study	Mean age ≥55	RCT	Physical illness	Validated outcome measure	Mean base-line depression measure	depression as focus of treatment and outcome.	CBT?
Teri, L., Logsdon, R. G., Uomoto, J., & McCurry, S. M. (1997). Behavioral treatment of depression in dementia patients: a controlled clinical trial. <i>The journals of gerontology. Series B, Psychological sciences and social sciences</i> , 52(4), P159-66.	✓	✓(76.4)	✓	✓	✓HDRS/BDI	✓HDRS: 15.4/BDI 17.9	✓ focus on depression	✓ BT/PST approach adapted, dementia
Pibermik-Okanovic, M., Begic, D., Ajdukovic, D., Andrijasevic, N., & Metelko, Z. (2009). Psychoeducation versus treatment as usual in diabetic patients with subthreshold depression: preliminary results of a randomized controlled trial. <i>Trials</i> , 10, 78. Vuk Vrhovac University Clinic, Dugi dol 4a, Zagreb, Croatia. pibermik@idb.hr.	✓	✓(55)	✓	✓	✓CES-D	✓CES-D median 26	✓	✓ CBT psych-ed for depression
Moorey, S., Cort, E., Kapari, M., Monroe, B., Hansford, P., Mannix, K., Henderson, M., et al. (2009). A cluster randomized controlled trial of cognitive behaviour therapy for common mental disorders in patients with advanced cancer. <i>Psychological medicine</i> , 39(5), 713-23. doi:10.1017/S0033291708004169	✓	✓(64)	✓	✓	✓HADS	9.91-10.78	✓	✓
Lustman, P. J., Griffith, L. S., Freedland, K. E., Kissel, S. S., & Clouse, R. E. (1998). Cognitive Behavior Therapy for Depression in Type 2 Diabetes Mellitus. <i>Internal Medicine</i> , 613-621.	✓	✓(54.8 = 55)	✓	✓	✓BDI	21-25	✓	✓

Appendix 13: Assessment of Risk of Bias: Authors' Consensus (Meta-Analysis).



Authors (Date)	Bias	Authors' judgement of risk	Support for judgement
<b>Dao et al. (2011)</b>	Random sequence generation (selection bias) Allocation concealment (selection bias) Blinding of outcome assessment (detection bias) Incomplete outcome data (attrition bias) Selective reporting (reporting bias) Other bias	Low Unclear Unclear Low Unclear Low	'Individuals who scored 14 or greater on the BD-II or 40 or greater on the STAI were randomly assigned using a random numbers table to receive the TAU or a briefform of CBT.' e110.N Method of allocation concealment not described. Outcome measures both self-report STAI-Trait, and BDI. No blind clinician assessment undertaken.  1 person died. Only one person in each group dropped out, although reasons not given, not likely to bias results. No intention to treat analysis No protocol, so not able to discern risk of selective reporting bias. No power calculations and limited follow up, limit validity of conclusions with regard generalizing and long-term effects. AD use examined and NS. Therapist training and monitoring of adherence to tx modality and use of a purpose designed manual for tx. Good flowchart, increase transparency
<b>Dobkin et al. (2011)</b>	Random sequence generation (selection bias)  Allocation concealment (selection bias) Blinding of outcome assessment (detection bias)  Incomplete outcome data (attrition bias) Selective reporting (reporting bias) Other bias	Low  Low Low Unclear Low	'Appropriate candidates were allocated to receive CBT plus clinical monitoring or clinical monitoring only (1:1 ratio) by computer-generated random assignment (run by the statistical consultant [M.A.G.]). Randomization was stratified by antidepressant use at screening (yes/no) and conducted in blocks of six consecutive participants within each stratum'. p1067 Undertaken by computer-generated random assignment. see above. Transparent reporting of blinding tests and measures taken to re-blind if broken by patient: Although kappa coefficient .48; moderate accuracy in guessing; this guess tested against HAM-D change over time and not related p=.62. Intention to treat analysis used and relatively low drop-out with reasons given. Full trial protocol accessed by contacting the author: not found as pre-published. 1. 'Patients were recruited from the Richard E. Heikkila Movement Disorders Clinic, local newspapers and the New Jersey Chapter of the American Parkinson's Disease Association between April 2007 and March 2010.' and 2. Inclusion criteria: 'had a family member or friend willing to participate.' risk of non-representative sample bias.
<b>Foley et al. (2010)</b>	Random sequence generation (selection bias) Allocation concealment (selection bias) Blinding of outcome assessment (detection bias)  Incomplete outcome data (attrition bias) Selective reporting (reporting bias) Other bias	Low Low Low  Low Unclear Unclear	'On the basis of a computer-generated allocation sequence, sealed, opaque envelopes detailing treatment allocation were prepared by an individual who was not involved in the study' See above Blinding reported and tested: 'Consistent ratings allowed the clinicians to proceed independently.' All assessments were done within a week of their target. The blindness of raters was tested: One rater guessed participant condition with 47.5% accuracy, and the other rater guessed it with 52.5% accuracy. These results were not meaningfully different from the 50% expected by chance' 8 withdrew: MBCT= 2 'unwell' Control: 1 died 2 'unwell' 3 'withdrew'. ITT analysis (3% attrition) more DO in control but missing data strategies discussed for ITT analysis (used LOCF) No protocol, so not able to discern risk of selective reporting bias. Source of participants via advertisements and inclusion criteria very broad. Potential impact of AD meds not reported, but therapist training and monitoring of adherence to treatment modality and use of manual for treatment.

Authors (Date)	Bias	Authors' judgement of risk	Support for judgement
Freedland <i>et al.</i> (2009)	Random sequence generation (selection bias)	Low	'We used a SAS program (SAS Institute, Cary, North Carolina) to generate a random allocation sequence with block sizes of 3 and 6.'
	Allocation concealment (selection bias)	Unclear	'Group assignments were concealed in sealed envelopes and revealed to the study coordinator immediately after the participant completed all of the baseline assessments'. no mention of opacity or sequential numbering.
	Blinding of outcome assessment (detection bias)	Unclear	'outcome assessors were masked to the patient's baseline data and group assignment' integrity not tested
	Incomplete outcome data (attrition bias)	Low	'conformed to the intention to treat principle' using imputed data: 1/41 CBT, 5/42 SSM, 3/40 UC.
	Selective reporting (reporting bias) Other bias	Unclear Low	No protocol, so not able to discern risk of selective reporting bias. Integrity of intervention and training of therapists well documented
Geillis <i>et al.</i> (2008)	Random sequence generation (selection bias)	Low	'Randomization was blocked so that assignment to the intervention and control conditions was equalized after every eighth assignment using a computer-generated random allocation table.' p.598
	Allocation concealment (selection bias)	Low	see above
	Blinding of outcome assessment (detection bias)	Unclear	'Trained interviewers were blind to study assignment and completed follow-up interviews at posttreatment, 3 months, and 6 months'. 'Raters who were blinded to the participant's intervention condition and not involved in patient assignment or treatment scored the instruments' Blinding reported but integrity not tested
	Incomplete outcome data (attrition bias)	High	7/69 attrition. No ITT Although 'A nonparametric analysis comparing dropouts from completers found no significant differences on age, gender, or pre-test scale scores', this does not account for possible selective drop-out.
	Selective reporting (reporting bias) Other bias	Unclear High	No protocol No test of fidelity of interventions, power calculation or assessment of AD use.
Hynninen <i>et al.</i> (2010)	Random sequence generation (selection bias)	Unclear	'include d patients were divided into matched pairs according to their post-pronchodilatory FEV1 predicted, and from each pair one participant was randomly assigned to CBT and the other to enhanced standard care' matched pair design likely to reduce effect of baseline imbalances, but no information about how randomly assigned.
	Allocation concealment (selection bias)	Unclear	'Allocation concealment was implemented using numbered containers that were identical in appearance for the two groups' not clear that they were opaque
	Blinding of outcome assessment (detection bias)	Unclear	Outcome measures both self-report BAI BDI-II. No blind clinician assessment undertaken.
	Incomplete outcome data (attrition bias)	Low	ITAU had greater drop-out 18/26 completed follow up as compared with 23/25 but intention to treat analysis undertaken
	Selective reporting (reporting bias) Other bias	Unclear High	Pre-published protocol not found. Relatively small and underpowered, Source described (partially via advertisements) not likely to be representative. No attendance/fidelity scores

Authors (Date)	Bias	Authors' judgement of risk	Support for judgement
Koertge et al., (2008)	Random sequence generation (selection bias)	Unclear	'A person not in contact with patients allocated them to either the intervention or to the control group by means of a chance table'. Randomly assigned by chance table but some pre-screening as asked to participate only if they were able to attend all 20 tx sessions. This will bias recruitment but may do so for both groups. This appears to have removed 140 potential participants.
	Allocation concealment (selection bias)	Unclear	'The result of the randomization procedure was kept in sealed envelopes and was given to the patients by research nurses. The person entering patients' data in the computer had no knowledge about the study,' not clear that they were opaque
	Blinding of outcome assessment (detection bias)	Unclear	Self-rated measures: blinding not possible. 'subjective, non-blinded measurement of the self-rated vital exhaustion and depressive symptoms might have biased our results'.
	Incomplete outcome data (attrition bias)	Unclear	Number for attrition given but not reasons for not completing questionnaires
	Selective reporting (reporting bias)	Unclear	Pre-published protocol not found
	Other bias	High	1. 'patients in the treatment group were in the care of a cardiologist, whilst the patients in the control group were treated as usual, i.e. they were more likely to have been under the care of a general practitioner. This difference is likely to have resulted in differential treatment with regard to types of medication, dosage and number of visits.' 2. Fidelity of intervention not tested
Kunik et al. (2008)	Random sequence generation (selection bias)	Low	'We used the Statistical Analysis Systems PLAN procedure (SAS Institute, Inc., Cary, NC, USA) to create the randomization list, with blocks of size 2 to provide approximately equal numbers per class.
	Allocation concealment (selection bias)	Unclear	'The statistician provided randomization numbers and treatment codes to the study coordinator when sufficient patients for two classes had completed the baseline assessment and consented to participate. The instructor assigned the treatment to the code initially by flipping a coin'. Not clear treatment codes and randomization numbers were already twinned when handed over: coin toss could introduce bias.
	Blinding of outcome assessment (detection bias)	Unclear	'Study personnel performing assessments were blinded to treatment condition' integrity not tested.
	Incomplete outcome data (attrition bias)	Unclear	56/120 attrition for education group 52/118 for CBT group. Intention to treat undertaken but very high drop-out reasons for drop-out between groups not clear.
	Selective reporting (reporting bias)	Unclear	Pre-published protocol not found
	Other bias	High	mixed methods including flyers /adverts for recruitment. mainly white male veterans & 40% of patients did not meet criteria for DSM-IV diagnoses limiting its generalisability. Psychotropic COPD medication changes not monitored.
Lincoln & Flannaghan (2003)	Random sequence generation (selection bias)	Low	A computer-generated random number sequence was prepared in advance and sealed in opaque, consecutively numbered envelopes by an independent researcher. The random allocation was not stratified, as there was no prior information on variables likely to affect outcome' 112
	Allocation concealment (selection bias)	Low	See above
	Blinding of outcome assessment (detection bias)	Unclear	'Outcome assessments were administered by an assistant psychologist, who was blind to the group allocation, 3 and 6 months after randomization' 112 blind not tested.
	Incomplete outcome data (attrition bias)	Unclear	Less than 5% attrition at end of treatment without skew, but 8.9% at FU with more in CBT group 'refusing' No protocol, so not able to discern risk of selective reporting bias.
	Selective reporting (reporting bias)	Unclear	Assessment of compliance with experimental treatments not undertaken and training of clinician limited: risk of poor fidelity to CBT treatment. AD use reported and potential impact assessed.
	Other bias	High	

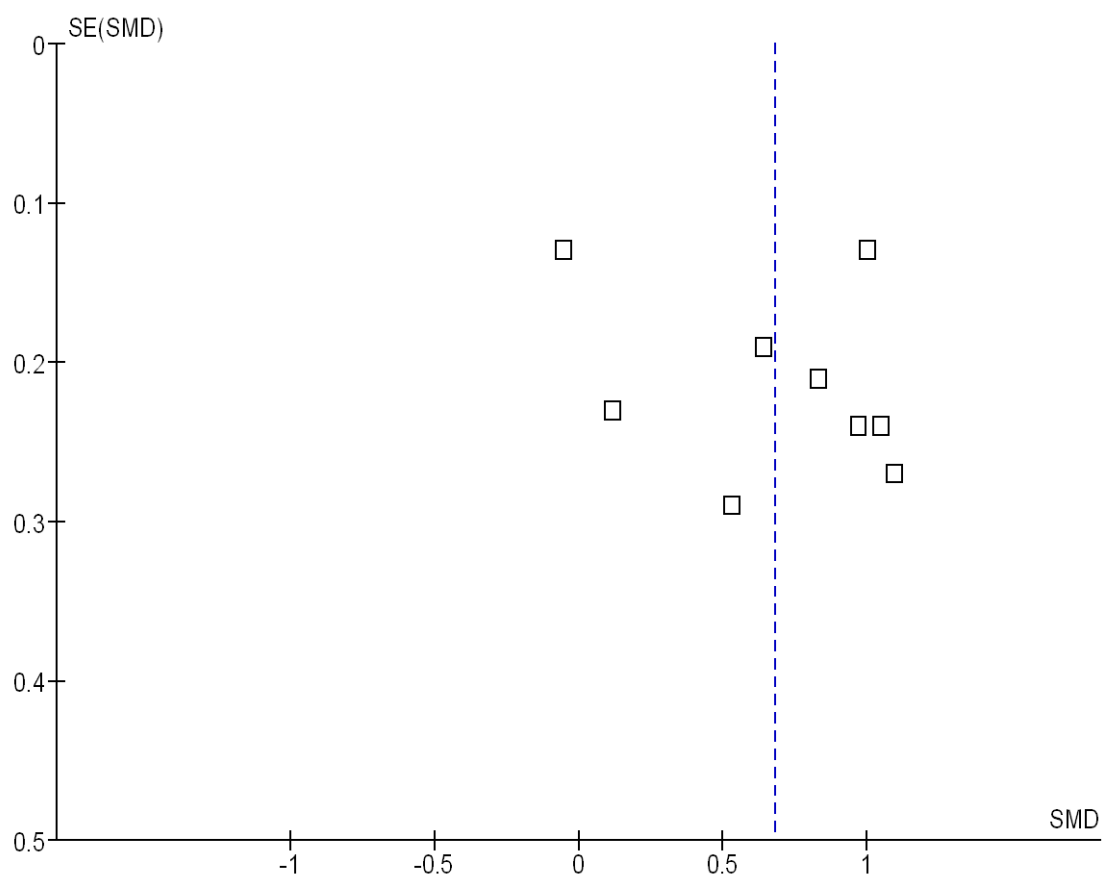
Appendix 14: Quality Rating of Included Papers: Authors' Consensus (Meta-Analysis).

Consensus scores on Moncrieff et al., (2001) Quality Rating Scale (QRS)

Moncrieff, J. Churchill, R., Drummond, D.C., McGuire, H. (2001) Development of a Quality Assessment Instrument for Trials of Treatments for Depression and Neurosis. *International Journal of Methods in Psychiatric Research*, 10, (3), 126-133.

	Lincoln & Flannaghan (2003)	Gellis et al., (2007)	Koertge et al., (2008)	Freedland et al., (2009)	Foley et al., (2010)	Hynninen et al., (2010)	Dobkin et al., (2011)	Dao et al., (2011)
1	1	2	1	2	1	1	2	1
2	2	0	2	2	2	2	0	2
3	2	2	2	2	2	1	2	0
4	2	0	0	2	2	0	2	2
5	2	2	2	2	2	2	1	2
6	2	2	2	2	2	2	2	2
7	1	2	1	2	2	2	2	2
8	0	0	0	0	0	0	0	0
9	2	1	1	2	1	2	1	1
10	2	2	0	2	2	1	2	2
11	2	1	1	2	1	2	2	2
12	1	2	2	2	2	2	1	2
13	1	1	1	1	1	2	0	2
14	0	0	0	2	2	1	0	2
15	0	0	0	0	0	0	0	0
16	2	2	1	2	2	2	1	2
17	2	2	2	2	2	2	2	2
18	2	2	2	2	1	2	2	2
19	0	0	2	2	2	0	2	2
20	1	2	2	2	1	2	2	2
21	2	2	2	2	2	2	2	2
22	2	2	2	2	2	2	2	1
23	2	2	2	2	2	0	2	2
<b>Totals</b>	<b>33</b>	<b>31</b>	<b>30</b>	<b>39</b>	<b>38</b>	<b>30</b>	<b>32</b>	<b>39</b>

Appendix 15: Funnel plot of standard error (SE) against effect size (SMD) (Meta-Analysis).



Funnel plot of standard error (SE) against effect size (SMD) from data comparing CBT with treatment as usual or waiting list control for all studies at end of therapy (patient-rated depression outcome measures were used in preference to clinician-rated outcomes when available).