

Review

# Problem solving therapies for depression: A meta-analysis

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## Abstract

**Purpose.** – In the past decades, the effects of problem-solving therapy (PST) for depression have been examined in several randomized controlled studies. However, until now no meta-analysis has tried to integrate the results of these studies.

**Methods.** – We conducted a systematic literature search and identified 13 randomized studies examining the effects of PST, with a total of 1133 subjects. The quality of studies varied.

**Results.** – The mean standardized effect size was 0.34 in the fixed effects model and 0.83 in the random effects model, with very high heterogeneity. Subgroup analyses indicated significantly lower effects for individual interventions in studies with subjects who met criteria for major depression, studies in which intention-to-treat analyses were conducted instead of completers-only analyses, and studies with pill placebo and care-as-usual control groups. Heterogeneity was high, and the subgroup analyses did not result in clear indications of what caused this high heterogeneity. This indicates that PST has varying effects on depression, and that it is not known to date what determines whether PST has larger of smaller effects.

**Conclusion.** – Although there is no doubt that PST can be an effective treatment for depression, more research is needed to ascertain the conditions and subjects in which these positive effects are realized.

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**Keywords:** Problem-solving therapy; Depression; Meta-analysis

## 1. Introduction

In the past few decades, dozens of psychological treatments have been developed for the treatment of depression [7,9,25]. However, only a limited number of these treatments have been examined in well-designed randomized controlled trials, and only very few therapies have been examined in five or more trials [24]. Most research has been conducted on cognitive behavior therapy and to a lesser extent on interpersonal therapy, couple and marital therapy, and life review therapy for older adults [24,8].

Problem-solving therapy (PST) is another psychological treatment which has been examined in several randomized controlled trials. In PST, the patient systematically identifies

his or her problems, generates alternative solutions for each problem, selects the best solution, develops and conducts a plan, and evaluates whether this has solved the problem [20,11].

There are several types of PST for depression. The first type, ‘social problem-solving therapy’ (SPST) was developed in 1980s [11,22], and is typically conducted in a group format of 10–12 sessions. This treatment does not only focus on the problem-solving skills themselves, but also on changing those attitudes or beliefs that may inhibit or interfere with attempts to engage in the remaining problem-solving tasks. The second type, PST for primary care (PST-PC), was developed in 1990s [20], and is applied individually in six sessions. It focuses on the core elements of problem-solving and can be used by trained nurses. The third type of problem-solving, self-examination therapy (SET) [5,6], is aimed at determining the major goals in their life, investing energy only in those problems that are related to what matters and learning to accept those situations that

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cannot be changed. Problem-solving skills are the core element of this approach. SET is typically used in a guided-self-help format, but can also be applied in group and individual settings.

Early studies on PST in 1980s used volunteer samples recruited from the community [21,23], while more recent studies have examined the effects of PST in primary care [20] and clinical settings [1]. However, until now no systematic review or meta-analysis has attempted to integrate the results of randomized trials of PST. This is especially important because some studies have found strong effects of PST [21], while others found none or only very modest effects [26].

While several trials of PST have reported positive results, we wanted to examine whether these results remain significant in a meta-analytic approach and we decided to conduct a comprehensive meta-analysis of randomized controlled trials of PST.

## 2. Method

### 2.1. Identification and selection of studies

Studies were traced by means of several methods. First, we used a large database of 777 papers on the psychological treatment of depression in general. This database was developed through a comprehensive literature search (from 1966 to March 2005) in which we examined 5178 abstracts in Pubmed (1224 abstracts), Psycinfo (1336), Embase (1118) and the Cochrane Central Register of Controlled Trials (1500). We identified these abstracts by combining terms indicative of psychological treatment (psychotherapy, psychological treatment, cognitive therapy, behavior therapy, interpersonal therapy, reminiscence, life review) and depression (both MeSH-terms and textwords). For this database, we also collected the primary studies from 22 meta-analyses of psychological treatment of depression [8]. For the current study, we examined the abstracts of these 777 studies, and selected the ones which focused on PST. In addition, we examined the references of earlier reviews on PST [24,18], and we reviewed the reference lists of retrieved papers.

We included studies in which (1) the effects of PST (2) on adults (3) with a depressive disorder or an elevated level of depressive symptomatology (4), were compared to a control condition or another (psychological or pharmacological) treatment (5) in a randomized controlled trial. No language restrictions were applied.

We defined PST as a psychological intervention in which the following elements had to be included: definition of personal problems, generation of multiple solutions to each problem, selection of the best solution, the working out of a systematic plan for this solution, and evaluation as to whether the solution has resolved the problem.

### 2.2. Quality assessment

There are at least 25 scales available to assess the validity and quality of randomized controlled trials [14]. There is no evidence, however, that these scales provide more reliable

assessments of validity. We preferred therefore to use a simple approach for assessing the validity of the studies, as suggested in the Cochrane Handbook [14]. We assessed the validity of the studies using four basic criteria [14]: allocation to conditions is done by an independent (third) party; adequacy of random allocation concealment to respondents; blinding of assessors of outcomes; and completeness of follow-up data.

### 2.3. Meta-analysis

We calculated effect sizes ( $d$ ) by subtracting (at post-test) the average score of the control group ( $M_c$ ) from the average score of the experimental group ( $M_e$ ) and dividing the result by the average of the standard deviations of the experimental and control group ( $SD_{ec}$ ). An effect size of 0.5 thus indicates that the mean of the experimental group is half a standard deviation larger than the mean of the control group. Effect sizes of 0.56–1.2 can be assumed to be large, while effect sizes of 0.33–0.55 are moderate, and effect sizes of 0–0.32 are small [15].

In the calculations of effect sizes we only used those instruments that explicitly measure depression (Table 1), such as the Beck Depression Inventory [4], and the Hamilton Depression Scale [12]. If more than one depression measure was used, the mean of the effect sizes was calculated, so that each study (or contrast group) only had one effect size. In one study more than one experimental condition was compared to a control condition [23]. In this case, the number of subjects in the control condition was evenly divided over the experimental conditions so that each subject was used only once in the meta-analyses.

To calculate pooled mean effect sizes, we used the computer program Comprehensive Meta-analysis (version 2.2.021), developed for support in meta-analysis. As we expected considerable heterogeneity, we decided to calculate mean effect sizes both with the random effects model and the fixed effects model. In the fixed effect model it is assumed that all studies in the meta-analysis are drawn from the same ‘population’ of studies and all factors which could influence the effect size are the same in all the study populations. In the fixed effects model the observed effect size differs between studies only because of the random error inherent in each study. In the random effects model, on the other hand, it is assumed that the included studies are drawn from ‘populations’ of studies that differ from each other systematically. In this model, the effect sizes resulting from included studies differ because of the random error within studies (as in the fixed effects model), but also because of true variation in effect size from one study to the next.

In our analyses, we have tested whether there are genuine differences underlying the results of the studies (heterogeneity), or whether the variation in findings is compatible with chance alone (homogeneity) [13]. As indicator of homogeneity, we calculated the  $Q$ -statistic. A significant  $Q$  rejects the null-hypothesis of homogeneity and indicates that the variability among the effect sizes is greater than what is likely to have resulted from subject-level sampling error alone. We also calculated the  $I^2$ -statistic which is an indicator of heterogeneity

in percentages as well. A value of 0% indicates no observed heterogeneity, and larger values show increasing heterogeneity, with 25% as low, 50% as moderate, and 75% as high heterogeneity [13].

We examined whether the effect sizes of specific subgroups differed from each other, with the methods for subgroup analyses as implemented in Comprehensive Meta-analysis version 2.2.021.

### 3. Results

#### 3.1. Description of studies

Thirteen studies, with a total of 1133 subjects (588 in the PST conditions, and 545 in the control conditions) met the inclusion criteria and were included in the current study [5,6,23,1–3,26,10,17,16,19–21]. Selected characteristics of the included studies are described in Table 1. In six studies, subjects were recruited from the community, while in the other seven, subjects were recruited from primary care or clinical settings. Three studies were aimed at older adults, and the remaining nine studies focused on younger adults. In seven studies, the participating subjects had to meet diagnostic criteria for a major depression. The remaining six studies included subjects who scored high on a self-rating depression, or also included subjects who met criteria for other mood disorders. In eight studies PST was delivered in individual format, in four studies in group format, and in one study as guided self-help. The number of sessions in the individual and group interventions varied between six and 12. Several different types of control groups were used: waiting list (four studies); care-as-usual (three studies), pill placebo (three studies), and psychological placebo intervention (two studies); while one study did not include a control condition (but compared PST to other treatments). In all studies, effect sizes could be directly calculated from the reported means and standard deviations, so that we did not have to use other statistics (*t*-value, *p*-value) to calculate effect sizes.

The quality of studies varied. Only three studies reported that allocation to conditions was conducted by an independent party. Concealment of random allocation to respondents was not possible or not reported in any of the studies, while blinding of assessors was reported in six studies. Drop-out numbers ranged from 0 to 42.6%. In five studies, intention-to-treat analyses were conducted; the other studies were limited to completers-only analyses.

#### 3.2. Effects of PST at post-test

We were able to compare the effects of the psychological treatments at post-test to control conditions in 12 studies with 13 contrast groups (Table 2), totalling 1053 subjects (508 in the PST conditions, and 545 in the control conditions). The mean effect size was 0.34 (95% CI: 0.02–0.48) in the fixed effects model and 0.83 (95% CI: 0.45–1.21) in the random effects model. Heterogeneity was high ( $Q = 69.7$ ;

$p < 0.001$ ;  $I^2 = 82.8\%$ ). We have plotted the effect sizes and 95% confidence intervals of the individual contrast groups in Fig. 1.

In eight studies (nine comparisons), the BDI was used as an outcome measure. In a meta-analysis in which only the effects of PST on the BDI were used, comparable results were found (fixed effects model:  $d = 0.52$ ; 95% CI: 0.35–0.70;  $Q = 38.3$ ;  $p < 0.001$ ;  $I^2 = 79.1\%$ ; random effects model:  $d = 1.05$ ; 95% CI: 0.54–1.55), as was the case when the results were limited to HRSD (seven comparisons; fixed effects model:  $d = 1.16$ ; 95% CI: 0.86–1.46;  $Q = 23.8$ ;  $p < 0.001$ ;  $I^2 = 74.8\%$ ; random effects model:  $d = 1.26$ ; 95% CI: 0.64–1.89).

The weight of three studies was large (one with a relative weight of 35.35% [10]; and two with a relative weight of 18.77% each [26,3]). We examined whether heterogeneity would be reduced if these studies were removed from the meta-analyses. However, these analyses resulted in comparable outcomes (Table 2).

#### 3.3. Subgroup analyses

Because of the high heterogeneity, we decided to conduct a series of subgroup analyses. Subgroups of studies analyzed included the following: age group (adults or elderly); diagnosis (only MDD; or other inclusion criteria); type of PST (SPST; PST-PC; and SET); recruitment (volunteers recruited from the community; or clinical/GP populations); format of the intervention (individual or group intervention); control group (waiting list; care-as-usual; placebo; psychological placebo condition); and type of analysis (intention-to-treat or completers only). The results of these analyses are presented in Table 2.

Several subgroups of studies resulted in significantly higher effect sizes: studies with group interventions had larger effect sizes than studies with individual interventions; studies aimed at subjects who met criteria for major depression had smaller effect sizes than studies in which other inclusion criteria were used; and the three studies in which intention-to-treat analyses were conducted had smaller effect sizes than the studies in which completers-only analyses were conducted. Furthermore, the type of control group was significantly related to the resulting effect size, with waiting list control groups having the highest mean effect size, and pill placebo the lowest mean effect size. And type of PST was also significantly related to the effect size, with the SPST having the largest effect size, and PST-PC having the smallest (non-significant) effect size.

Although several subgroups were significantly different from each other in effect size, the number of subgroups with low levels of heterogeneity was small. All subgroup analyses resulted in subsets of studies with  $I^2$  levels of higher than 50%, with the exception of three subgroups: the three studies with care-as-usual control groups ( $I^2 = 0$ ;  $d = 0.27$ ), the two studies with psychological placebo control groups ( $I^2 = 0$ ;  $d = 0.99$ ), and the two studies with self-examination therapy ( $I^2 = 0$ ;  $d = 0.94$ ). Because of the small number of studies and because of the relatively large number of

Table 1  
 Characteristics of randomized controlled trials examining the effects of problem solving treatment on depression

First author	Year	C	Target population			Conditions	N	Intervention				Meas	Instr	DO	ITT/ CO
			Age group	Recruitment	Diagnosis			T	N <sub>Sess</sub>	Time	Form				
Alexopoulos [1]	2003	US	Elderly, with executive dysfunction	Clinical	MDD (SCID) + HRSD $\geq$ 18	1. PST	12	1	12	Nr	IND	Pre, post	HRSD	0	ITT
						2. Control (Supp ther)	13								
Araon [2]	1993	US	Elderly	Community	MDD (RDC) + $\geq$ BDI + $\geq$ GDS $\geq$ 10 + HRSD $\geq$ 18	1. PST	28	1	12	1080	GRP	Pre, post, 3 mn	HRSD, BDI, GDS	10.7	CO
						2. Reminiscence	27								
						3. Waiting list	20								
Barrett [3]	1999	US	Adults (18–59)	Referrals from GP	DYSTH or minD + HRSD $\geq$ 10	1. PST	80	2	6	210	IND	Pre, post	HSCL-D, HRSD	20.0	ITT
						2. AD	80								
						3. Drug placebo	81								
Bowman [5]	1995	US	Adults	Community	HRSD $\geq$ 10	1. PST (SET)	10	3	4 tel. calls	20	BIB	Pre, post	HRSD, BDI	6.3	CO
						2. Cognitive MCP	10								
						3. Waiting list	10								
Bowman [6]	1996	US	Adults	Drug abuse inpatients	MMPI-D T-score $\geq$ 65, or MCMI-II D scale $\geq$ 75	1. PST (SET)	14	3	8	240	GRP	Pre, post	SCL-90-R-D	21.4	CO
						2. Current events grp	14								
Dowrick [10]	2000	EU	Adults (18–65)	Community survey	MDD, DYSTH, or adjustment disorder (SCAN)	1. PST	128	2	6	240	IND	Pre, post, 12 mn	BDI	25.6	ITT
						2. CBT	108								
						3. Care-as-usual	189								
Lynch [17]	1997	US	Adults	Primary care	SD (High MOS-D SI; no MDD/DIS)	1. PST	11	1	6	120	IND	Pre, post	HRSD, BDI	17.2	CO
						2. Care-as-usual	13								
Lynch [16]	2004	US	Adults ( $\geq$ 18)	Screening in GP practices	HRSD 11–26	1. PST	18	1	6 tel. calls	120	IND	Pre, post	BDI, DHP-D	42.6	CO
						2. Stress management	18								
						3. Care-as-usual	18								
Mynors-Wallis [20]	1995	UK	Adults (18–65)	Referrals from GP	MDD (RDC) + HRSD $\geq$ 13	1. PST	30	2	6	210	IND	Pre, post	HRSD, BDI	13.2	CO
						2. AD	31								
						3. Drug placebo	30								
Mynors-Wallis [19]	2000	UK	Adults (18–65)	Referrals from GP	MDD + HRSD $\geq$ 13	1. PST (GP)	39	2	6	210	IND	Pre, post, 12 mn	HRSD, BDI	23.2	ITT
						2. PST (pract. nurses)	41								





Table 2  
Meta-analyses of studies examining the effects of problem solving treatments on depression compared to control conditions at post-test: overall results and subgroup analyses

Study		<i>N</i> <sub>comp</sub>	<i>d</i>	95% CI	<i>Q</i>	<i>I</i> <sup>2</sup> (%)	<i>p</i> -Value
<i>Overall effects</i>							
All studies	Fixed effects	13	0.34	0.02–0.48	69.7 <sup>†</sup>	82.8	
	Random effects	13	0.83	0.45–1.21			
3 studies excluded <sup>a</sup>	Fixed effects	10	1.04	0.78–1.30	26.67***	66.3	
	Random effects	10	1.20	0.73–1.67			
BDI <sup>b</sup>	Fixed effects	9	0.52	0.35–0.70	38.30 <sup>†</sup>	79.1	
	Random effects	9	1.05	0.54–1.55			
HRSD <sup>c</sup>	Fixed effects	7	1.16	0.86–1.46	23.83***	74.8	
	Random effects	7	1.26	0.64–1.89			
<i>Subgroup analyses</i>							
Control group	Waiting list	5	1.61	1.19–2.04	12.57**	68.2	†
	Care-as-usual	3	0.27	0.06–0.48	0.94 ns	0	
	Placebo	3	0.05	–0.16–0.25	7.94**	74.8	
	Psychol. placebo	2	0.99	0.42–1.56	0.04 ns	0	
Age group	Adults	10	0.37	0.21–0.52	51.13 <sup>†</sup>	82.4	Ns
	Elderly	3	0.27	0.01–0.53	18.19 <sup>†</sup>	89.1	
Type of PST	SPST	7	1.20	0.86–1.54	24.46 <sup>†</sup>	75.5	†
	PST-PC	4	0.14	–0.01–0.29	9.69**	69.0	
	SET	2	0.94	0.35–1.54	0.00 ns	0	
Format	Individual	8	0.20	0.05–0.34	17.97**	61.0	†
	Group	5	1.56	1.15–1.97	13.38**	70.1	
Diagnosis	Only MDD	6	0.15	0.01–0.30	18.84***	73.46	†
	Other	6	1.27	0.95–1.60	18.84***	73.5	
Recruitment	Community	7	0.37	0.20–0.53	54.8 <sup>†</sup>	89.0	*
	Clinical/GP	6	0.30	0.08–0.53	14.74**	66.1	
Analyses	Intention to treat	4	0.12	–0.04–0.27	26.67***	70.0	†
	Completers only	9	1.04	0.77–1.31	26.67***	70.0	

\**p* < 0.1; \*\**p* < 0.05; \*\*\**p* < 0.01; and <sup>†</sup>*p* < 0.001.

<sup>a</sup> Barrett et al. [3], Dowrick et al. [10], Williams et al. [26].

<sup>b</sup> Only the effects on the BDI were included in this meta-analysis.

<sup>c</sup> Only the effects on the HRSD were included in this meta-analysis.

superior effects of control conditions) to huge, standardized effect sizes (*d* > 3). Several other characteristics of the included studies were found to be related to the effect size: format (group interventions had larger effects than individual interventions); diagnosis (studies including only subjects

with major depression had smaller effect sizes); type of PST (SPST having the largest and PST-PC having the smallest); type of analysis (intention-to-treat analyses resulted in smaller effect sizes); and type of control group (studies with waiting list control groups had the largest effect sizes).

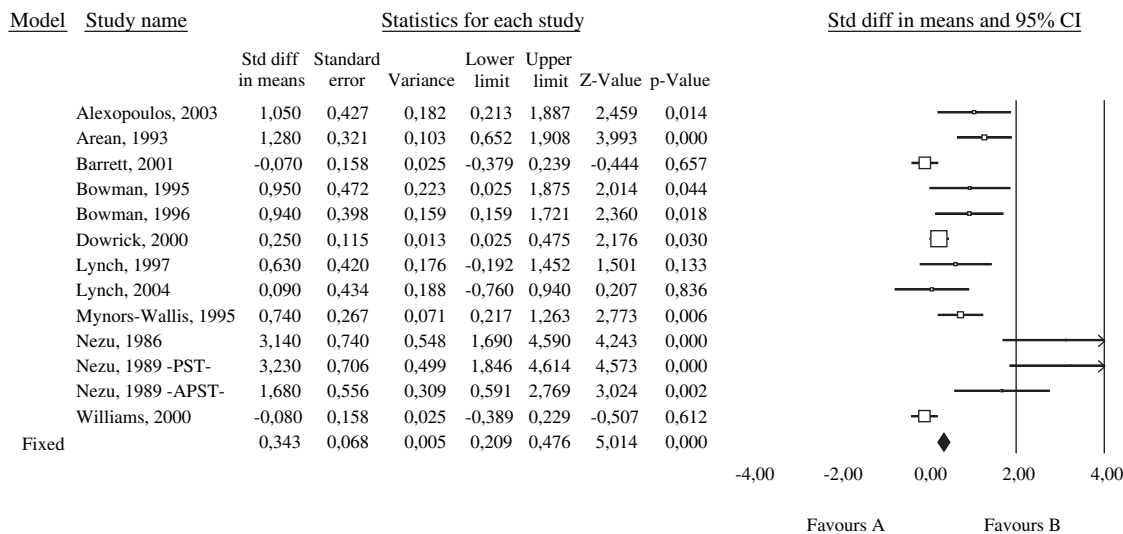


Fig. 1. Standardized effect sizes of problem solving treatment for depression compared to control conditions at post-test.

It was remarkable that PST for primary care (PST-PC) did not have a significant effect on depression ( $d = 0.14$ ) compared to control conditions in the studies we included in our meta-analysis.

However, heterogeneity was very high in almost all analyses. The subgroup analyses did not result in clear indications of what caused this high heterogeneity. This indicates that PST has varying effects on depression, and that it is not known to date what determines whether PST has larger or smaller effects.

Our subgroup analyses indicated that SPST is far more effective than PST-PC. This result, however, should be interpreted with caution, because heterogeneity is high in both subgroups of studies. This indicates that it is probably not the type of PST (SPST or PST-PC) that explains the large difference in effect size between these two subgroups. The studies on SPST and PST-PC differ on several important characteristics. For example, the majority of SPST studies drew from non-clinical participants from the community, had extremely small sample sizes, used only a waiting list control condition, and based their main outcome analyses on completers only. By comparison, the PST-PC studies are much stronger methodologically in these areas (i.e., sample drawn from patient populations, large sample sizes, placebo control and medication comparison conditions, and ITT analyses). Several of these same variables were shown through other subgroup analyses to be associated with outcome. Therefore, we cannot draw any firm conclusions about the potential superior efficacy of SPST over PST-PC.

This study has several limitations. First, we found that the quality of several studies on problem-solving as a treatment of depression was not optimal. Although it is clearly inherent in studies of psychological treatments that it is not possible to conceal to subjects to which condition they are assigned (in waiting list control conditions it is not possible at all), many studies did not meet other major quality criteria, such as assignment to conditions by an independent person, and blinding of assessors. Most studies only conducted completers-only analyses instead of intention-to-treat-analyses.

Another important limitation of this meta-analysis is that we were only able to include a relatively small number of studies. Furthermore, most studies used a waiting list or a care-as-usual control group, and very few studies used placebo or other control groups.

Although there is no doubt that PST can be an effective treatment for depression, more research is needed to ascertain the conditions and subjects in which these positive effects are realized.

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